A CONTINUED STUDY ON EPILEPSY- CLINICAL AND ELECTROENCEPHALOGRAPHIC ASPECTS.

THESIS

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C E R T I F I C A T E

This is to certify that the work entitled "A CONTINUED STUDY ON EPILEPSY - CLINICAL AND ELECTRO-ENCEPHALOGRAPHIC ASPECTS", which is being submitted as thesis for M.D.(Medicine) examination of Bundelkhand University, 1990 by Dr. PAWAN SOOD, has been carried out in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department according to the University regulations.

Dated: 11-9-, 1989.

(R. C. Arora)
M.D., D.Sc.,
Professor and Head,
Department of Medicine,
M.L.B. Medical College,
Jhansi.

C E R T I F I C A T E

This is to certify that the work entitled "A CONTINUED STUDY ON EPILEPSY - CLINICAL AND ELECTRO-ENCEPHALOGRAPHIC ASPECTS" has been carried out by DR. PAWAN SOOD under my direct supervision and guidance in the department of Medicine, M.L.B. Medical College, Jhansi. He has fulfilled necessary requirements of the stay in the department for the submission of thesis. The techniques, observations and calculations incorporated in this thesis have been undertaken by the candidate himself and checked by me from time to time.

Dated: 11-9-.1969.

(D. N. Mishra)
MD, MNAMS, FCCP,
Professor,
Department of Medicine,
M.L.B. Medical College,
Jhansi.

(GUIDE)

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Pawan Sood

(Pawan Sood)

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INTRODUCTION

INTRODUCTION

Epilepsy is a well known disease even to a layman. Epilepsy is having a long history and has also been mentioned in Ayurvedic literature. Most of the time epilepsy is a life long disease and it creates problem especially when children have to stop their studies or when the earning capacity of the adults are affected.

In the past most of the epilepsies were thought to be idiopathic in nature and no attempt was made to find out its curable cause.

It is easy to diagnose epilepsy on the basis of history especially when given by a person, who had seen an epileptic fit or on clinical examination just after attack. However, at times it is difficult to diagnose epilepsy in some particular situations, viz. when proper history could not be obtained or when the attack has not been seen by a person, convulsions in infant do not always means epilepsy. Attacks of grand mal associated with fever are referred to as febrile convulsions. In tetanus, attacks of grand mal are accepted as a integral part of the illness; when fits occur after a prolonged bout of coughing the use of the word epilepsy is not justifiable. However, if the convulsions alone are present without the above mentioned association than the diagnosis of epilepsy is made without any question. With the help of EEG one can differentiate epilepsy from hysterical fits most of the time but not

always. EEG might help in finding whether the epilepsy is generalized or focal or focal with secondary generalization. EEG can tell us the exact site of the discharge.

EEG often gives the vague idea in telling about the aetiology of the seizures. Such as if slowing is seen in the record than we can suspect that there is a destructive lesion in the brain like tumor. If spikes or sharp waves are seen than we suspect that there is a excitatory lesion like scar mark.

The diagnosis of epilepsy is made mainly on clinical grounds and EEG might be helpful to confirm the clinical diagnosis. In equivocal cases finding suggestive of epilepsy will be diagnostic. Normal EEG does not exclude epilepsy. Apart from EEG we can take the help of other investigations such as CT Scan, Positron Emission Tomography and Magnetic Resonance Imaging. The facilities of these investigations are not available in most of the centres.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL ASPECTS

Epilepsy has the longest medical history (Hoch and Knight, 1947). It was recognised in India before the tenth century B.C., and in the Greek literature as early as the fifth century B.C. (Joshi, 1973).

Various terms had been used - such as - the falling disease, fits, seizures and epilepsy. Famous person in the world like - Alexander the great, Julius Caesar, St. Paul Napolean, Lord Byron and Maupassant had fits (Lelemen, 1979), hence these attacks has also been known as 'sickness of the greates'. Galen (131-201 AD) described epilepsy as "the seizure of mind and sense together with a sudden fall in some with convulsions and in others without". The word 'aura' meaning 'breeze' was introduced by the teacher of Galen (Desai, 1968).

Four hundred years B.C., Hippocrates named it as "The sacred Disease". He clearly recognised that epilepsy had its seat in the brain. He gave a very precise and clear discription of what today would be called temporal lobe epilepsy or psychomotor seizures.

Epilepsy is defined as "Apasmara" in Indian Medicine, the prefix 'apa' meaning negation or less and 'smara' meaning recollection or consciousness. Charaka who lived in the second century B.C., described epilepsy

as "paraxysmal loss of consciousness due to disturbances of memory and understanding of mind attended with convulsive seizures". In the olden days many of the clinical features of the epilepsy were known. Aura was recognised and was called 'Apasmara Poorvaroopa'. Charaka, in his 'Nidana', gives list of symptoms which indicate the aura of the disease. Worthy of mention are subjective sensation of sounds, constriction sense in the chest, a sense of darkness, vertigo and dream - like state. Sushruta added to the discription of aura, a feeling of persistent flow of thought towards a particular topic which the patient is unable to control. In addition to the discription of the actual attack both of these authors mentioned mental symptoms occurring in epileptics (Ramamurthy and Gurunatham, 1969).

Aretaeus, in the second century described the various auras as hallucinations of vision, hearing and taste at the onset of a seizure. Erastus in 1580 also described the aura at the onset of the attack(Desai,1968).

Prichard (1822) stated that delerium could occur without a fit. Bright (1831) described an epileptic who had no convulsions. The term 'Aura intellectual' was introduced by Falret (1860) to describe various complex psychological phenomena. Association between the mental disorder and epilepsy had been recognised since the begining of the medical history.

DEFINITION OF EPILEPSY

Epilepsy is regarded as a state of 'Paraxysmal cerebral dysarrhythmia' (Gibbs et al, 1937). Penfield (1941) later wrote that one could regard epilepsy 'Physiologically as a tendency to periodic involuntary neuronal explosions!

Epilepsy is a paroxysmal and transitory disturbance of the functions of the brain which develops suddenly ceases spontaneously and exhibits a conspicious tendency to recurrence. The most acceptable definition of epilepsy given by Richter (1987) is "Epilepsies are a group of disorders characterised by chronic, recurrent paroxysmal changes in neurologic function caused by abnormalities in the electrical activity of the brain".

Migraine may present as a stereo typed, repeated transient neurological symptoms which may not be followed by headache, mimic a seizure. Patient of migraine may have abdominal pain and vomiting without headache. Vomiting may be the only feature of temporal lobe epilepsy (Shukla and Mishra, 1981).

EPIDEMIOLOGY

Epilepsy is a common melody, prevalence rate i.e. the total cases of epilepsy in a fixed population per thousand at a given time has been found to vary from as low as 2-4/thousand to as high as 10.5/thousand(Table 1).

According to the college of General Practitioner, 1960 epilepsy affects 5/thousand population.

Table 1

Showing prevalence of epilepsy in general population.

Author(s)	Place of study	Prevalence rate per thousand
Crombie et al (1960)	England and Wales	4.2
Brewir et al (1966)	Carlisle in Great Britain	5.5
Mathai (1971)	India	9.0
Grudzinska (1974)	Poland	3.4
Hauser and Kurland (1975)	U.S.A.	5.3
Juul-Jensen (1976)	Denmark	6.9
Fry (1982)	Kent	10.5
Hauser et al (1986)	U.S.A.	6.78

These variations in prevalence were because of different criteria for selection of cases, for example, inclusion of only active cases in some studies or inclusion of patients with febrile seizure or single seizure in other studies.

SEX AND AGE SPECIFIC PREVALENCE RATES FOR EPILEPSY

In the vast majority of studies quoted in table 1 males tend to predominate but in the study of Juul Jensen's and Hauser's studies there were more females and also in the Gover's study of 3000 cases the female : male ratio was 13:12. Several authors have suggested that the

high male rate is due to more frequent head injuries. The prevalence rates of post-traumatic epilepsy were estimated in the study female/male at 1.9 - 1.5 per thousand (Zielinski, 1977).

Age specific prevalence rates in several studies (Brew's et al, 1966; Grudzinska, 1974; Juul-Jansen, 1976; and Haerer et al, 1986) found to be showing almost similar pattern. The lowest rates, which usually occur in the first decade, increase in the older age groups and then show a marked drop after 50. Only in Haerer's (1986) study high prevalence rate was found in sixth decade. According to the office of health economics, 1971, the commonest age of onset is 0-4 years. The incidence of initial attacks then declines steadily throughout adult life with a further slight peak especially in males. Over the age of 65 years. Similar figures have been reported from many other countries including Switzerland, Holland and United States.

PATHOPHYSIOLOGY

The work of Li and Jasper (1961) has demonstrated clearly that the epileptic process consists fundamentally of hyperactive and hypersynchronous neuronal discharges, the abnormality of neurones appears to be in the instability of cell membranes. An inhibitory feed back mechanism similar to Renshaw cell system of the spinal cord probably exists in the brain (Eccles, 1965 and 1967) and is likely to play an important part in preventing the excessive

neuronal discharge that forms the basis of an epileptic attack (Phillip, 1959).

The bilaterally synchronous wave and spike cortical discharges which characterise petit mal attacks seem to originate sub cortically, perhaps in the interthalamic nuclei (Jasper et al, 1947) and the resulting impairment of consciousness was interpreted by Williams (1950) as indicating a blockade of different impulses to the cortex. In the light of experimental work and behaviour of EEG in man, Gastaut et al (1960) suggested that a grand mal seizure seemed to depend on a thalamic discharge, which involved the non specific reticular structures and was projected to the cortex in what might be considered a generalized recruiting response transmitted along the diffuse cortical projection pathway. For postictal paralysis Efron (1961) gave cogent reasons for suggesting that an active process of inhibition, resulting from persistent subclinical epileptic discharge, was a probable explanation.

Disturbances of consciousness, mood and behaviour which occurred as a result of discharges originating in the temporal lobe were thought to indicate a dysfunction played of that part of the brain (Penfield and Jasper, 1954).

Ward (1961) put forward evidences suggesting that epileptogenic foci were characterized by a standing negative potential of the order of 7-12 mv which could be attributed to a continuous state of dendritic depolarisation. Aimone (1961) emphasised that the discharge of the

primary epileptic neurones was only the begining of the process; before a clinical attack developed there had to be local recruitment of more and more neurones both locally and at a distance.

It has been shown in cat (Prince and Wilder, 1967) that the interictal discharges of an epileptogenic focus with their negative potentials are accompanied by intense inhibitory activity of the neurones in an extensive area of surrounding cortex. This is likely to be a factor in limiting the spread of the discharge.

Fundamentally, epilepsy is a physico-chemical disturbance, and the physico-chemical state of the neurones can be influenced by numerous agencies. Symonds (1959) suggested that the gamma - aminobutyric acid (GABA) might be a natural anticonvulsant formed in the brain. Local lesions might cause seizures either by allowing the local accumulation of an excitatory substance or by depressing the GABA concentration or the tonic inhibitory control of afferent impulses.

GABA and acetylcholine (Ach) have opposite effects upon neuronal excitability so that an imbalance between these two substances within the brain could be a factor predisposing to seizure production.

The balance between Ach and GABA may be upset for instance by pyridoxine deficiency as the latter substance is essential for the synthesis of GABA (Sutherland and Eadie, 1960) - Two receptor sites have been characterised

(Spero, 1982) - GABA/chloride - ionophor/benzodiazepine receptor - complex, and a specific phenytoin receptor.

Meldrum (1982), in reviewing pathophysiology of epilepsy, stressed the importance of metabolic factors like concentration of arterial PO_2, PCO_2, glucose, sodium, calcium, magnesium, urea and ammonia and the extent of change in serum osmolarity which have been associated with seizure activity in man.

Within the first 30 minutes after a generalised seizure activity, arterial hypertension, a rise in cerebral venous pressure, an increase in cerebral blood flow, hyperglycemia, hyperkalaemia, haemoconcentration and a low normal PO_2 with a high arterial PCO_2 are usual, while after 30 minutes there is often arterial hypotension, a raised or normal, cerebral venous pressure, a normal cerebral blood flow, hypoglycemia with persistent hyperkalaemia and mec hyperpyrexia (Meldrum, 1982). Prolactin LH and FSH may also rise post-ictally, the latter only in females (Dana et al, 1983). Low vitamin D and serum calcium levels are often found in patients with chronic epilepsy (Davis et al, 1983).

ETIOLOGY OF SEIZURES

Aetiology of the seizures depends upon the age of onset of the seizures. There are different causes operating in the neonates, infants, early childhood, childhood and adolescence, early adult life and late adult life.

According to the age group causes of epilepsy are as follows (Laidlaw and Richens, 1982).

1. Neonatal (1st month : Birth injury, birth anoxia, congenital abnormalities, metabolic disorders, meningitis and other infections.
2. Infancy (1-6 months) : As above and infantile spasms.
3. Early Childhood(6 months-3years) : Febrile fits, birth injury, infection, trauma, poisons and metabolic defects, cerebral degenerations.
4. Childhood and Adolescence : Idiopathic or primary epilepsy, birth injury, trauma, infection, cerebral degeneration.
5. Early adult life : Trauma, tumour, idiopathic or primary epilepsy, birth injury, infection, cerebral degeneration.
6. Late adult life : Vascular disease, trauma, tumour cerebral degeneration.

Now the common causes of epilepsy are discussed in brief.

Head Injury

Only a small proportion of head injured patients suffer from fits once they have recovered from the acute stage of injury. However, head injuries are so common in occurrence that this comprises a sizable number of patients Fits due to head injury may develop soon after the injury or months later. The risks of epilepsy are related to

whether the duramater is penetrated or not. In a study (Caveness et al, 1962) incidence of convulsions were found to be 40% in those who suffered from missile wounds of the head. While only about 5% of those with non-missile head injuries developed seizures (Jenett, 1962 and 1965). Trauma is more likely to cause partial than generalised epilepsy and may be responsible for fits in about 5-15% of all cases of epilepsy (Gibbs and Gibbs, 1952). Epilepsy after head injury is divided into early or late epilepsy. Jennet (1962) proposed that definition of early epilepsy should be reserved for the fits in the first week after injury. Patients who develop fits in first two weeks after injury, some 27% will continue to have persistent recurrent seizures, but in those whose fits develop after two weeks or later, risk is of order of 70% (Jennet, 1969). Jennet et al (1973) have above to identify factors likely to be associated with persistent post traumatic epilepsy after head injury. In investigation of 900 patients with epilepsy after non-missile injuries there were over 400 with early epilepsy, over 400 with late and 90 with both (Laidlaw, 1982). It is late epilepsy which is usually meant when the term traumatic epilepsy is used, because it is this which constitutes persisting disability. In a study (Annegers et al, 1980) of 2747 patients of mild head injury, incidence of seizures was not significantly greater than in general population.

Brain Tumour

Brain tumour is considered to be an important cause of late onset of epilepsy from a very long period (Parker, 1930 and Penfield et al, 1940). In fact brain tumours are responsible for later onset epilepsy only in about 10% of all cases (Shehan, 1958; Raynor et al, 1959; Hyllsted and Pakkenberg, 1963; Juul, 1964a). Incidence of tumours rises steeply in the cases of partial fits, where a figure of 30-40% is more appropriate (Raynor et al,1959; Sumi and Teardall, 1963). However, the incidence in complex partial seizures is not that high being about 15% (Currie et al, 1971). Meningiomas and benign gliomas characteristically cause seizures, while malignant gliomas do so less frequently. The incidence of fits in 67% in meningiomas, 70% in astrocytomas, and 37% in malignant gliomas (Penfield et al, 1940). Secondary deposits from lung and breast is also a common cause of fits (Laidlaw, Richens, 1982). Fits are commonest in tumours of fronto-parietal region and very rare with lesion in thalamus, basal ganglia or parapituitary area (Williams, 1965). Tumour as a cause of fit in children is uncommon, partly because of most childhood tumours arise in non-epilepto-genic areas such as the cerebellum, brain stem and diencephalous and also because so many other epilepsy appears in childhood.

Local cerebral lesion - other than tumours are also responsible for epilepsy. Among the many lesions

described at autopsy have been chronic localised encephalitides (Rasmussen et al, 1958), focal cortical dysplegia (Taylor et al, 1971), neuronal heteretopias hamartomas, meningioangiomatosis, and other vascular malformations (Matheson, 1992; Leblanc et al, 1983). Arteriovenous malformation causes fits usually focal in nature in about 40% of cases (Peterson et al, 1956). Fits are said to be rare in subdural haematoma.

Cerebrovascular diseases - is an even more common cause of adult onset fits than tumour (Dodge et al, 1954). It may be responsible for 10-20% cases of adult onset epilepsy, but after the age of 50 the figure is 50% or more (Juul-Jensen, 1964a and Wardcock and Cobgrove, 1964). It is estimated that as many as 25% of those with cortical infarcts will have fits (Richardson and Dodge, 1954), although the incidence of fits in non-embolic cerebral infarcts in general is about 8%(Louis and McDowell, 1967).

Migraine - while loss of consciousness in an attack of migraine, often at the height of the headache, is usually syncopal, there is a slightly increased incidence of epilepsy in migraine sufferers even in those who have no evidence of a cerebral lesion. A possible role of tyramine in the physiological mechanism of epilepsy and migraine was postulated by Scott et al, (1972).

Perinatal asphyxia - has been found to be a very common cause of epilepsy. In a study of Brown et al(1974)

out of 94 infants who suffered from asphyxia, 32 had birth injury and 48 had convulsions.

Hereditary predisposition - plays a considerable part in idiopathic epilepsy. EEG abnormalities are six times more common in relatives of epileptics than the controls (Lennox et al, 1949). Lennox (1947) believed that the dysarrhythmia is inherited as a mendalian dominant trait although the predisposition is clearly of relatively low penetrance (Brown, 1982).

CLINICAL CLASSIFICATION OF SEIZURES

The terminology and classification of seizures has evolved over many years, creating a variety of interchangeable and confused descriptive terms.

Traditionally, attacks of epilepsy, whether idiopathic or symptomatic have been divided into major epilepsy (Grand mal), minor epilepsy (Petit mal), focal epilepsy (Jacksonian epilepsy), temporal lobe epilepsy (psychomotor epilepsy) and myoclonic attacks (Janz, 1969). However, this descriptive classification has become increasingly unsatisfactory for many reasons. Thus there are many forms of minor epilepsy with or without transient impairment of consciousness, which are not true petit mal; in epilepsy of focal onset, depending upon the site of origin in the brain, a variety of motor, sensory, behavioural and psychomotor manifestation may be found but if the epileptic discharge spreads rapidly to become generalised, a major attack may occur and the focal symptoms

then constitute merely the aura of the major attack. Temporal lobe epilepsy is now more generally known as complex partial epilepsy (Penry and Daly, 1975).

Many new classifications have been proposed notably by the International League Against Epilepsy (Gastaut, 1969); Sutherland and Eadie (1980) and the comprehensive clinical and electroencephalographic classification recommended by the International League Against Epilepsy, the World Federation of Neurology and the International Federation of Societies for Electroencephalography and Clinical Neurophysiology.

A simpler working classification, from Marsden and Reynolds (1982) is given as follows. They point out that it is and always will be, impossible to create a single code to cover three basically incompatible systems of classification viz one according to the clinical signs and symptoms in the attack; one relating to the anatomical and physiological evidence as to its source; and one defining aetiology.

Classification of Epilepsies

(Marsden and Reynolds, 1982)

I. Generalised

- Tonic-clonic
- Tonic
- Atonic
- Absence

- Atypical absence
- Myoclonic

II. Partial (Focal)

A. Without impairment of consciousness (Simple partial Seizures).

B. With impairment of consciousness (Complex partial Seizures).

i) With motor signs (e.g. Jacksonian Versive).

ii) With somato or special sensory symptoms (e.g. Olfactory, visual).

iii) With autonomic features (e.g. epigastric sensations).

iv) With psychic symptoms (e.g. fear).

v. With automatism(complex partial Seizures only).

III. Partial Seizures Secondarily Generalised

Clinical or electrical evidence of focal discharge during or after the generalised seizures.

IV. Unclassifiable

Seizures which cannot be classified because of incomplete data.

PHENOMENOLOGY

GENERALISED SEIZURES

One of the most common type of epileptic attack is the generalised seizures. Some of these appear to be primary generalised seizures and others are the result of

secondary generalisation of partial seizures.

Tonic Clonic grand mal seizures

The classical attack of epilepsy consists of preconvulsive symptoms, aura, convulsion and the post-convulsive phase. In pre-convulsive symptoms, some patients experience a warning signal for hours or even a day or two. These vague symptoms include irritability and depression, abnormal feelings referred to the head, giddiness and sudden myoclonic twitches. In other cases patient had no warning but becomes unconscious at once. Aura is less common in major seizures than in the seizures of focal onset.

Primary generalised seizures as described by Richter (1987) usually start without warning, although some individuals sense a vague nonspecific sense of the impending event. The onset is heralded by sudden loss of consciousness, a tonic contraction of the muscles, a loss of postural control, and a cry produced by a forced expiration caused by contraction of the respiratory muscles. The individual falls to the floor in an opisthotonic posture, often sustaining injury and remains rigid for many seconds. There may be cyanosis as respiration is inhibited. Soon a series of rhythmic contractions of all four limbs occur. The clonic phase can last for a variable period of time and ends when the muscles relax. The individual remains conscious and unarousable for a period of minutes or longer. There is usually a gradual return to consciousness and often there is a period of disorientation

during recovery. Post-ictally, Headache and drowsiness are common sequelae and the individual may not return to baseline functioning for days.

The clinical features of a major fit in a child are similar to those in the adult. However, children often do not go through the postictal phase of the coma, confusion, headache and sleep, but usually recover completely within minutes (Laidlaw and Richens, 1982).

Tonic Seizures

Tonic seizures are a less common form of generalised seizures which consist of spasm of the limbs or torso often with deviation of the head and eyes towards one side (Dichter, 1967). They are not followed by clonic phase and are often of shorter duration; usually 10-20 seconds (Gastaut et al, 1963).

Absence (Petit mal) Seizures

Petit mal is now defined as brief absence of attacks, occurring almost always in childhood (6-14 years of age) and associated with characteristic EEG paroxysms of three per second, bilateral, syndronous spike and wave discharge (Gibbs et al, 1935). It rarely appears for the child losses his consciousness, his eyes stare, and he may show minor movements such as blinking or twitching of the face and arms but he does not fall. Suddenly consciousness is regained but there is total amnesia during the brief period of the attack. The patient looks around for a

moment but then resumes his previous occupation. If engaged in conversation before the attack, he will have missed a sentence or so (Laidlaw and Richens, 1982). Such classical petit mal attacks may occur very frequently even as often as hundred or more times daily (Lennox, 1945; Gibberd, 1966; Dalby, 1969). Automatic minor motor phenomenon may resemble complex partial seizures (Penry, 1975). Other possible associated clinical features of absence seizures include myoclonus, version of the head and conjugate deviation of the eyes, decreased postural tone and autonomic changes. Approximately 50% of patients with absence seizures also experience generalised tonic-clonic seizures (Gibberg, 1966 and Lugaresi, 1973). Absence status may last for hours, with clouding of consciousness and reduced accuracy of responses (Manual, 1963). There is usually no period of post ictal confusion. Status of petit mal does not appear to have any deleterious prognostic implications (Andermann, 1972).

The onset of absence is rare before the age of three years; it is most common between four and eleven years of age, and unusual after age of twenty years (Andermann, 1972).

Atypical Absence Seizures

The association of the EEG pattern of 'Petit mal variant" (Gibbs, 1971) or "Slow spike wave"(Lennox, 1949) with certain clinical aspects led to the concept of a syndrome variously named 'severe myokinetic epilepsy of

early childhood with slow spike and wave" (Sorel, 1964). Childhood epileptic encephalopathy with diffuse slow spike waves" (Gastaut, 1966), Lennox Syndrome (Schneider, 1970) and "Lennox-Gastaut Syndrome (Heidermeyer, 1969; Gastaut, 1979). The term Lennox-Gastaut Syndrome was proposed by Gastaut et al (1966) to designate a form of epilepsy of childhood with frequently repeated fits of several types and an interictal EEG pattern of diffuse spike - waves at a rhythm of approximately 2 Hz, previously described as petit mal variant (Gibbs and Gibbs, 1952). The role played by genetic factors is controversial. The incidence of family history of convulsive disorder is low (2.5%) in some series (Chvrie and Aicardi, 1972) and very high(50%) in other series (Doose et al, 1970). Mental retardation is present from the onset in 20-60% of patients and in half of the cases it is severe. The prognosis of Lennox Gastaut Syndrome is poor. According to Gastaut (1973), 80% will continue to have seizure. It is characterised by onset of seizures early in childhood. More than one variety of generalized seizures occur; predominantly tonic tonic-clonic, atonic, akinetic, absence and myoclonic (Chenrie, 1972; and Blume, 1973). Mental retardation is often present. The EEG contains generalized sharp and slow wave complexes (Markand, 1977). The maximal age expression of the Lennox-Gastaut syndrome is between 1 and 5 years with a slight preponderance of males (Scolio-Levissari,1977).

During 'minor' status epilepticus i.e. the prolonged episodes of frequently repeated akinetic or myoclonic seizures, the patient may be unable to maintain the head erect. The accompanying difficulty in eating and swallowing resembles pseudobulbar palsy (Doose, 1970). Delayed psychomotor development has been found in 20-30% of patients (Blume, 1973). Additional abnormalities in the neurological or ophthalmological examination are found in 30-59% of patients (Markand, 1977). Mental defecit was especially prevalent in patients who had infantile spasms, tonic seizures or minor status epilepticus (Blume, 1973).

Atonic Seizures

Atonic seizures are brief losses of consciousness and postural tone not associated with tonic muscular contractions. The individual may simply drop to the floor without apparent cause. Atonic seizures usually occur in children and are often accompanied by other forms of seizures. The EEG contains polyspikes and slow waves. The "drop attack" of atonic seizure needs to be distinguished from cataplexy seen in narcolepsy(where the patient remains conscious); transient brain stem ischaemia; or sudden rise in intracranial pressure (Richter, 1987).

Sudden falls have also been described in patients with partial epilepsies especially partial complex seizures (Caffi, 1973 and Delgado, 1982). Stress had been given upon the serious nature of partial epilepsies with drop attack because they often resist drug treatment (Roger, 1981 and Poole, 1985).

Myoclonic Seizures

Myoclonic seizures are sudden brief, single or repititive muscular contractions envolving one body part or the entire body, in which seizure is accompanied by a violent fall without a loss of consciousness. Myoclonic phenomena is a very common experience and epilepsy is only one of their many causes. Many people experience myoclonic jerkings when falling asleep. Myoclonic attacks that are epileptic in nature have been regarded as fractionated or miniature attacks of grand mal (Gastaut and Fischer-Williams, 1959b). Marsden et al (1979) pointed out that myoclonus is used as a descriptive clinical term which has no physiological, aetiological or therapeutic implications. Myoclonic epilepsy of adolescence usually begins at about puberty (Jeavons, 1977), rarely before the age of 9 years, with myoclonic jerks involving the head, arms and upper trunk.

Infantile Spasm or Hypsarrhythmia

This form of primary generalised seizure occurs in infants before one year of age. Peak age is 3 and 8 months (Jeavons, 1964). It consists of brief synchronous contractions of the neck, torso and both arms (usually in flexion) lasting 2-10 seconds. Infantile spasms often occur in children with underlying neurologic disease, such as anemia encephalopathy or tuberous sclerosis, but can rarely occur in an otherwise normal infant. The prognosis of children with this type of seizure disorder is grave and approximately 90% develop mental retardation in

additions to their seizures. The EEG is characterized by a very disorganised background, random high voltage slow waves, spikes and burst suppression (hypsarrhythmia). The spasms and hypsarrhythmia tend to disappear over the first of generalized seizures. Psychomotor development may have been delayed before the onset of the infantile spasm, but more often the retardation is noted after their onset.

The association of infantile spasms, arrest of psychomotor developments and hypsarrhythmia has been called the West's syndrome (Gastaut, 1971).

PARTIAL SEIZURES

Simple Partial Seizures

Simple partial seizures can occur with motor sensory, autonomic or psychic symptoms without impairment of consciousness.

The typical partial motor fits consists of onset of tonic spasm followed soon by repetitive twitching, usually in the angle of mouth, thumb and index finger, or great toe which then spreads in an orderly manner. The convulsive movements may remain confined to their site of onset or may spread to involve one half of the body and may terminate in a typical grand mal fit with loss of consciousness. If the left hemisphere is the source speech may be lost during the attack (Laidlaw and Richens, 1982). Very rarely true inhibition of movement has been described as an epileptic event (Efron, 1961). Fits may be followed by Todd's paralysis. In patients, especially

in adults, a prolonged Todd's paralysis may be due to tumour causing the fit and in such cases if it persists for more than 48 hours, full investigation is required. Another not uncommon form of motor seizure is the adversive attack due to discharges arising in pre-motor areas of the frontal lobe (Penfield and Welch, 1951). Typically the head and eyes are forced away from the affected hemisphere, usually with preservation of consciousness.

Somatic sensory seizures characteristically commence in one of the preferred sites, such as thumb or mouth and show a spread of march. Complaint is usually of numbness or pins and needles usually motor phenomenon is associated.

Visual fits are due to discharges in an occipital pole and usually consist of unformed simple visual phenomena such as spots, flashes of light balls on fire throughout the visual field. Other types of visual seizures which are more common, are associated with temporal lobe epilepsy.

Auditory, gustatory and olfactory symptoms are usually found with temporal lobe seizure.

Visceral aura, consisting of abnormal feelings in epigastrium may be accompanied by autonomic phenomena, such as tachycardia, sweating, pupillary dilatation and a fall in blood pressure (Von Buren and Ajmonemarsen, 1960). Whether this sequence of autonomic events occurring briefly and repeatedly, but without any other evidence of epilepsy, is a form of restricted autonomic epilepsy, is

debatable, but has been found to respond to antiepileptic drugs (Fox et al, 1973).

Involuntary micturition can occur in isolation as a possible manifestation of seizure discharge, often with loss of awareness such that patient finds himself wet without prior knowledge of events (Maurice-William, 1974).

Complex Partial Seizures

Complex partial seizures are episodic changes in behaviour in which an individual losses conscious contact with the environment. The onset of these seizures consists of variety of aures.

These are more commonly known as psychomotor seizures. The term psychomotor epilepsy was first used by Van Gieson (1902) and again by Gibbs, Gibbs and Lennox (1937). Lennox (1951) suggested the name "temporal lobe epilepsy". Psychomotor epilepsy denotes clinical psychomotor attacks, irrespective of the presence and location of a focus while the term temporal lobe epilepsy means epilepsy with a temporal lobe focus in the EEG respective of the clinical seizure pattern (Stevens, 1966). Gibbs and Gibbs (1964) reported 80% incidence of temporal lobe foci on EEG of his patients with clinical psychomotor epilepsy. As against this typical psychomotor seizures may be associated with frontal epileptic foci (Penfield and Kristiansen, 1951). The reported incidence of temporal lobe epilepsy within the total epileptic population varies from 25% (Chaddy et al, 1966), 26%(Penfield

and Jasper, 1954), 34% (Zeilinski, 1974), 40% (Gastaut et al, 1975), 2.7%(Bagadia et al, 1973), 17.5% (Mahan, 1974) and 25% (Gopalakrishanan et al, 1968).

Majority of the patients had the onset of seizure below the age of 20 years (Shukla et al, 1978), while Gibbs et al (1948) had reported only 9.3% of the epileptics below age of 20 years. Currie et al (1971) reported maximum incidence of seizures in the third and fourth decades. However, 62-75% of cases of Aird et al (1967), Viramani and Sawhney (1966) and that of Reddy (1971) had their first attack in first two decades. Phenomenology of complex partial seizures is quite varied. More than one type of clinical seizure pattern may be exhibited by a patient of temporal lobe epilepsy (Falconer and Taylor, 1970).

1. Auras only.
2. Absences.
3. Psychomotor attacks.
4. Falling attacks.
5. Grand mal attacks.

It is not possible here to discuss every group separately and in detail. Fear or anxiety is the most common premonitory emotional experience (Williams, 1956 and 1968) and but some times the attack may cause pleasure of considerable intensity, as was described by Dostoievsky (1955). The onset of these seizures may consist of any of the variety of aura. Aura has been found in 60% of cases (Shukla et al, 1979). The commonest were visual halluci-

nation followed by vertigo and epigastric sensation. Unusual smell (as of burning rubber); a feeling that the current experience had happened before (dejavi); a sudden intense emotional feeling; a sensory illusion such as micropsia or macropsia or specially formed sensory hallucinations, could be other type of aura. Paroxysmal vomiting as a sole manifestation of temporal lobe epilepsy has also been reported (Shukla and Mishra, 1981).

Olfactory and gustatory auras once said to be diagnostic of temporal lobe seizures, are in fact rare (De Jong, 1957 and Dennerll, 1964).

A curious feature is the tendency of turn towards religion (Esquirol, 1838; Morel, 1860; Howden, 1872-73; Bowen, 1919; Slater et al, 1963 and Slater and Roth, 1969). It occurs most commonly in temporal lobe epilepsy, because in them the aura often takes the form of some religious experience (Karagulla and Robertson, 1955; Slater et al, 1963 and Sedman and Hopkinson, 1966). Dewhurst and Beard (1970) reported 6 cases of temporal lobe epilepsy with "religious conversion experiences".

During complex partial seizures there may be a cessation of ongoing activity with some new motor manifestations, which usually follow aura. Though epileptic attack can cease just after aura, sometimes generalised tonic-clonic seizures may be the final event in which usually there is amnesia for the aura.

Among the clinical features of temporal lobe seizures, several authors include "somatomotor manifestation"; localised or generalised, tonic, clonic, tonic-

clonic or postural manifestations (Lennox, 1960). In a study (Rossi, 1984) 10% of psychomotor epileptics had motor symptoms; lateral deviation of head and eye occur frequently (King and Ajmone-Marsan, 1977). Hypotonia may occur causing the patient to fall down(Geier et al, 1977).

Additional motor activity may be in the form of automatism which occur in the form of smacking of lips; running at the onset of seizure (Chen and Prostar, 1973). This can be in association of gelastic epilepsy, i.e. excessive laughter (Gumpert et al, 1976); Picking at clothes walking aimlessly. Sometimes, complex acts like motor driving or performing on musical instruments is automatic motor phenomenon; laughing or crying can occur (Offen et al, 1976). Sexual automatism in the form of masturbation or pelvic thrusting has been reported (Spencer et al, 1983). A significantly greater number of temporal lobe epileptics do have emotional disturbances in childhood and psychiatric abnormalities in later part of life, in comparison to patients with grand mal epileptics (Shukla et al, 1979).

Abnormal sexual behaviour associated with temporal lobe epilepsy has frequently been described in man (Gastaut and Colomb, 1954; Marchini and Zinisi, 1987 and Heirons and Saunders, 1966) and in animals(Kluver and Bucy, 1939). Almost all the types of sexual perversion have been observed in association with temporal lobe epilepsy.

The occurrence of hypersexuality in association with temporal lobe epilepsy is rather rare(Taylor, 1966b). However, Blumer (1969) reported 7 cases of temporal lobe epilepsy who had distinct episodes of hypersexuality, usually following abrupt cessation of seizure activity. Only one patient out of 100 in the series of Taylor(1969b) was hypersexual. A large number of temporal lobe epileptics were found to be hyposexual (Shukla et al, 1979).

Secondarily Generalisation of Partial Seizures

Simple or partial complex seizures can progress to get generalised with loss of consciousness and often with convulsive motor activity. This may occur immediately or after many seconds or a minute or two. In addition, many patients with focal seizures have generalised seizures without an obvious initial focal component and are difficult to distinguish from primary generalised seizures. The presence of an aura or the observation of any focal feature at the onset of generalised seizures or the presence of postictal focal neurological defecit (Todd's paralysis) are important clues to the focal origin of seizure (Richter, 1987).

Evoked or Reflex Epilepsy

Sometimes an attack can be excited by some form of external stimulation. This may be a sudden loud noise (acousticomotor epilepsy) or music (musicogenic epilepsy) or a visual stimuli e.g. reading (reading epilepsy)

(Binjel, 1957; Stoupel, 1968) or viewing television (Television epilepsy) (Brooks and Lirauch, 1971). In some cases attacks are precipitated by speaking and writing (language induced epilepsy) (Geschwind and Sherwin, 1967) or by blinking when starting to speak (Terzano, Parrino, Mansoni and Mancia, 1983).

INTERICTAL BEHAVIOUR IN EPILEPTICS

The association of specific behavioural changes with epilepsy has been questioned for long time (Freud, 1950; Lennox, 1940; Geschwind et al, 1980 and Reynolds, 1983). Community surveys in unselected populations have confirmed an increased incidence of psychological disturbances in epileptics (Rutter et al, 1960 and Gudmundsen, 1966). Recently use of objective measures of mental/ intellectual function, has been tried to document whether characteristic changes in cognitive or behavioural function occur in patients with epilepsy (Dodrill, 1978 and Giordani et al, 1985).

Dennis et al (1986) found a significantly low level of intellectual functions in patients with secondary generalised tonic-clonic seizure in comparison to other epileptic population. It appears that for newly diagnosed unmedicated epilepsy patients, the level of intellectual function compares favourably with that of general population. Lower levels of intellectual performance have frequently been reported for epilepsy samples (Kleve and

Mathews, 1969; Dodrill et al, 1974 and Reynolds, 1983). This may have resulted, in part, from inclusion of more chronic, poorly controlled and highly medicated epilepsy patients.

Even with the normal levels of intellectual performance for the epilepsy sample and the close age and education match between the control and epilepsy groups, significant comparative differences were consistently observed between the two group(Laidlaw and Richens, 1982).

EPILEPSY AND PSYCOSIS

Many authors have, however, failed to find any difference between the incidence of psychosis in different types of epilepsy (Alstrom, 1950; Vislie and Henriksen, 1958; Guerrant et al, 1962; Small et al, 1962; Juul-Jensen, 1964; Stevens, 1966 and 1973). Guerrant and coworkers (1962) evaluated 32 patients with psychomotor epilepsy, 26 with grand mall and 26 control cases with chronic medical illness, not involving brain. The study was designed to test the hypothesis that functional psychiatric disturbances were more common in psychomotor epilepsy. The hypothesis was not confirmed. Standage (1973) studied 53 patients of epilepsy in a mental hospital. Of these, 8 were psychotic.

LABORATORY INVESTIGATIONS

In a patient in whom we are suspecting epilepsy our aim is to confirm its diagnosis and then to decide its cause. The initial step in investigation of the case of epilepsy is a thorough history both from the patient and a witness and general and systemic examination. A number of causes can be recognised in the clinic itself. A history of birth trauma, or anoxia combined with body asymmetry, such as small thumb or toe will point out to cerebral damage in early life. Tuberous sclerosis and Sturge Weber syndrome will be indicated by the appropriate skin lesions and congenital or genetically determined syndromes may be suggested by a characteristic facial appearance or other signs i.e. mongolism. In some cases examination is directly concerned with detecting signs of focal brain damage or raised intracranial pressure.

INITIAL OUT PATIENT INVESTIGATIONS

Initially simple tests are required. In all patients, X-ray skull and EEG are required. Now various tests had been done such as routine blood count, VDRL test for syphilis and biochemical test for blood sugar and calcium. Even though one can rarely detect the cause of fit by these tests, however, these base line investigations should be performed in all the cases. X-ray chest is a necessary to exclude bronchogenic carcinoma (Laidlaw and Richens, 1982). Evidence of tuberculosis can also be seen by it. In some patients an X-ray of the

thigh may show the calcified cysts of cysticercosis. In idiopathic epilepsy the CSF is normal except that during or after frequent fits of an attack of status there may be a rise in pressure. A consistently raised CSF proteins and a pleocytosis should suggest that the epilepsy is symptomatic. Usually the X-ray skull is normal in the patients but at times it may show calcification in tumor, evidence of raised intracranial pressure or other lesions and bony changes of meningioma may be detected. Pineal shift may be the indicative of a mass lesion. Asymmetry, particularly of middle cranial fossa may point to a long standing atrophic lesion. Such informations are crucial in a few cases and a skull X-ray should be undertaken in every epileptic patient (Laidlaw and Richens, 1982).

ELECTROENCEPHALOGRAPHY

Historical aspects - In 1914 Cybulski recorded an epileptic seizure caused by cortical stimulation in a dog. All the earlier works were done on animals and it was not untill 1929 that Hans Burger published the first report of the electro-encephalogram of man. Pen writers were available in 1940's and made it possible to have an immediate permanent record. The other great technical advance at this time was the use of the differential amplifier which eliminated much of the interference from external sources (Mathews, 1934; 1938; Tunnicliffe, 1938; Parr and Walter, 1943). Since 1940, there have been only little change of basic technique.

INTERICTAL ELECTROENCEPHALOGRAPHY

It is seldom that a satisfactory EEG recording is obtained during the attack of fits because of the muscle and movement artefacts that occur. It is unusual for a routine EEG recording to coincide with an actual seizure and it is necessary therefore to depend on interseizure pattern for diagnostic assistance in most cases of epilepsy.

The diagnostic role of interictal EEG in epilepsy has been questioned by many authors (Goodin, 1984). Diagnosis of epilepsy is mainly clinical (Kellaway, 1981). Spike and wave and similar epileptiform discharges are sometimes found in the EEG of individuals who are not known to have epileptic attacks of any kind, emphasizing that epilepsy must always be diagnosed on clinical grounds and not solely on EEG evidence. Zavin and Ajmone Marsen(1968) found that of 6497 non epileptic patients examined at the National Institutes of Health, Bathesda, 142 (2.2%) had spikes or sharp waves with or without associated slow waves in their EEG. Thus the role of EEG is restricted to classify the seizure disorders, localising the epileptogenic focus and guiding prognosis (Mathews, 1964 and Critchley, 1978). In a study by Goodin (1984) in general population, 60% of epileptic patients had positive epileptic form of activity. On the other hand only 4% of non-epileptic had positive EEG. In another group who were suspected to have epilepsy, 93% positive EEGs belong to epileptic group. Absence of epileptiform activity in

EEG reduces the chances for epilepsy in the suspected cases from 50% (clinically) to 33%.

INTERSEIZURE EEG IN GENERALIZED EPILEPSY

Interseizure epileptiform discharges in generalised epilepsy are always widespread, bilaterally synchroneus and more or less symmetrical. The degree of abnormality of interseizure record is often related to the frequency of attacks and the time lapsed after the last attack.

Abnormalities found in EEG, in generalized epilepsy, are of many varieties. They are usually non specific but in some conditions are specific. In generalised tonic-clonic epilepsy various abnormalities are found like spike and wave, polyspikes, sharp waves, slow waves or sharp and slow wave complexes, but they must essentially be bilaterally synchroneous and almost symmetrical. Absence seizure show typical EEG pattern i.e. bilateral spike and wave activity of three hurts (Perry, 1975). These bursts show high degree of bilateral voltage symmetry, spatial distribution and synchrony.

Kellaway (1982) showed the relationship of age specific epileptiform EEG patterns to clinical seizures in children. Interictal EEG recording of patients with infantile spasm is almost always severely abnormal and typically demonstrates the abnormality known as hypsarrhythmia (Gibbs et al, 1952). This pattern rarely persists beyond the age of 4-5 years.

Various authors used different criteria to define Lennox-Gastaut syndrome. Some workers selected the EEG pattern as main criterion (Blume, David and Gomez,1973; Niedermeyer, 1969; Markand, 1977) and included all the clinical correlations of the diffuse slow spike wave pattern. Others base their discription mainly on a clinical picture domiated by falls and massive myoclonias (Kruse, 1968; Doose et al, 1970) and use the clinically descriptive term of 'Myoclonic-astatic petit mal' which has often and wrongly been considered synonymous with that of the Lennox Gastaut Syndrome. Atill others (Aicardi, 1973; Gastaut, 1973; Jeavons, 1977) used combined electro-encephalographic and clinical criteria.

Drop attacks or akinetic seizures are associated with various types of interictal EEG abnormalities like diffuse slow spikes - wave discharges; with or without focal epileptic abnormalities (Pacto, 1985). EEG accompaniment of myoclonic attack often resembles the spike and wave phenomena in so far as they consist of an association of high voltage spikes and slow waves which are bilaterally synchronous, but the episode seldom have the regularity of true spike and wave. In epileptic subjects these myoclonic discharges may be provoked by sudden noises or photic stimulation (Kiloh, 1981).

INTERSEIZURE PATTERNS IN PARTIAL EPILEPSY

The important features of interictal EEG in partial epilepsy is the presence of localised spikes and with a bipolar montage have a focal origin of which is usually seen in the form of phase reversals. They are usually monophasic but may be biphasic or triphasic. The duration of these waves is less than 80 milliseconds. In many patients the duration of discharge lies between 80 - 200 milliseconds and it is customary for these to be called as sharp waves. Sometimes the discharge originates in one hemisphere and is reflected as a mirror focus in the appropriate area of contralateral cortex; this is especially seen in frontal discharges and those originating from the medial aspects of the temporal lobe, owing to the richness of the commisural connections existing between homologous areas in the two hemispheres (Kiloh et al,1982). Ogden et al (1956) have shown that discharges are so closely synchronised that transcallosal connections cannot account for them. Morphology of the EEG activity in partial seizures may be sequential spikes and sharp waves (Jasper, 1949; Gastaut, 1953; Kiloh et al, 1972; and Klass, 1975). Although a spike or sharp wave focus provides the most frequent EEG evidence of a cortical epileptogenic lesion, the discharge may comprise spikes and slow waves of paroxysmal activity in alpha, theta, or delta ranges. Matthew and Knott (1977) pointed out that sharp waves are less reliable indicators of a cortical epileptogenic lesion than spikes. At times multiple independent foci

called as 'shifting foci' are seen. Migratory foci are particularly found in children. The initial focus may be superseded by another elsewhere.

The incidence of epileptiform abnormality is greater in epileptic children than in epileptic adults because there is tendency of a epileptic focus to move forward or tendency to diminish in frequency or disappear with increasing age, as seen by improvement in EEG (Gibbs and Gibbs, 1953).

In temporal lobe epilepsy, the medial aspect of the temporal lobe is the common site for epileptogenic foci due to its susceptibility to anoxic damage either at birth or as a result of febrile illness and because the threshold to stimulation of this region is the lowest of any area of cortex. When the site of origin of epileptiform discharge is on the convexity of one of the temporal lobe its discovery and localisation are easy (Engle et al, 1975). Deep temporal lobe lesion can given rise to distant spike. In the series of temporal lobe epilepsy, reported by Jasper, Pertuisset and Flanigin (1951) the focus was unilateral in 34%, bilaterally synchronous but consistently asymmetrical in 24%, bilaterally synchronous and of equal voltage in 19%, whilst in remaining 23% bilateral independent foci were present.

When a localised lesion gives rise to bilaterally synchronous and often symmetrical discharge the phenomena is referred to as secondary bilateral synchrony or

secondary generalization. Jasper (1952) and Penfield et al (1954) showed that it was possible for bilaterally synchronous abnormalities to appear in the EEGs of the patients with epilepsy caused by unilateral parasagittal, orbitofrontal or anterior temporal lesions.

PROVOCATIVE TECHNIQUES

Clinically significant changes in the EEG may be evoked by certain procedures as hyperventilation, photic stimulation and sleep.

1. Hyperventilation

Here patient is asked to take deep breaths at 20 respiratory rate for 2-4 minutes. The usual response to hyperventilation is the appearance of bilateral slow activity, due to the fall in arterial carbon dioxide. This response is potentiated by mild hypoglycemia occurring after three hours without eating. Changes are seen more in temporal lobe epilepsy and in children.

2. Photic Stimulation

This procedure activates the epileptogenic lesions in the occipital region evoking focal sharp waves or slow waves or both. In this technique repititive flashes of light are presented at rates ranging from 15-30 per second. The finding is rare since lesions in the occipital lobe are infrequent.

3. Sleep

In drowsiness and the early stages of sleep, generalised seizure discharges are sometimes enhanced. Niedermeyer (1955; 1966) claimed that patients with generalised seizure often show massive irregular bursts of spikes which are associated with K complexes. Pharmacological provocation have little or no importance because they can provoke epileptiform activity in normal individuals. The enthusiasm of Gibbs and his coworkers (1950) for sleep as a method of provocation is not shared by all, but its value is greatest in cases of temporal lobe epilepsy. Merlis, Greenman and Henriksen (1951) found that about half their patients with psychomotor epilepsy showed focal epileptiform discharges during sleep, as compared with about one third in the alert state, but Gloor, Tsai and Haddad (1958) pointed out that in only 7% of their patients sleep recording was necessary to obtain EEG confirmation of their clinical diagnosis of temporal lobe epilepsy. On the other hand Silverman and Morisaki (1958) considered that sleep recording was crucial in confir-ming the diagnosis in almost 20% of their mixed epileptic group. A more recent study by Niedermeyer and Rowan (1972) found that 23(31%) of 73 patients showed epileptic discharges only during sleep, whilst in another 33(45%) of these were enhanced. As a rule the patient of suspected temporal lobe epilepsy who has a nonspecific routine EEG and who does not fall to sleep spontaneously, should have a sleep recording carried out before being subjected to

the greater ordeals of the insertion of special electrodes. Any hypnotic may be used. Sleep recordings are usually obtained by depriving the patient of sleep for 24 hours (Pratt et al, 1969). Kriebel and Schlager (1973) used this technique in patients suspected of having seizures. Of routine EEG 8.6 per cent showed focal epileptiform discharges, while of the sleep recording,18 per cent showed such discharges. These mostly occurred as the patient was falling asleep. Scollo Lavizzari, Pratt and de La Cruz (1975) found that 138 of 294 epileptic patients whose routine records lacked supporting evidence, showed epileptiform discharges after sleep deprivation for about 24 hours. A significant increase in spike wave activity has been observed by Burr et al (1996) just after falling asleep and (less pronounced) after awakening in the morning. Halasz (1994) observed that micro arousals are especially related to the production of the spike wave paroxysms.

LUMBAR PUNCTURE

This procedure gives no useful information in a usual case of epilepsy. It is required when it is suspected that the epilepsy may be due to infection, encephalitis, or stroke or other intracranial disorders.

CONTRAST METHODS

Contrast methods are commonly used for the cases of computerised transaxial tomography(CT), carotid angiography pneumoencephalography and ventriculography.

a. CAROTID ANGIOGRAPHY

In this method we inject an iodine containing contrast media in common carotid artery. Here we can visualise cerebral arteries and their branches.

b. PNEUMOENCEPHALOGRAPHY AND VENTRICULOGRAPHY

Injection of air into a lumbar subarachnoid space in the patient with sitting position permits visualization in considerable detail of the size and position of the ventricles and subarachnoid space (upper spinal and cerebral) and indirectly the structures which lie between the ventricle and meninges.

Except CT scanning, other contrast methods are not used now a days as they are invasive procedures and less informative than CT scan(Boltshauer et al, 1977).

Computerized axial Tomography CT Scan)

It is an harmless procedure which is now being used widely. It differentiates epidural, subdural and intracerebral haemorrhages and deformities of the ventricular system from mass lesions and demonstrates tumours, abscesses, granulomas when done after an intravenous injection of meglumine diatrizoate (Renografin), or other contrast medium, as well as areas of brain oedema and infarction, hydrocephalous and brain atrophy. Gastaut (1976) has summarised the CT scan findings in 1702 epileptic patients of all ages combined from seven research groups. Over all proportion of abnormalities varied from

34-51% with a mean of 46%. Amongst these lesions 56% were atrophic in character. Tumours were found in 3-11%. The relationship of CT abnormalities to various seizure types is well illustrated by the study of Gastaut and Gastaut (1976) in 401 patients. A clear difference emerges between the relatively low incidence 11% associated with primary generalized seizures and the much higher proportion 60-80% in relation to other seizure types. These authors also estimated that CT scan detects 20% more cerebral lesions than the combination of long established techniques (Skull X-ray, EEG, angiography etc). These observations have been extended by Yang et al (1979) who scanned 256 children upto age of 17 years with a mean age of 4 years. Abnormalities were seen in 33%. They were able to distinguish a low yeild group (2.5 - 8%) with idiopathic generalised seizures. Simple partial seizures either solitary or recurrent, were more likely to be associated with structural disease (Yang et al, 1983; and Boodanoff, 1975). In study by Yang et al (1983) approximately half of the patients with simple partial seizures had positive scans and even a solitary focal seizure was likely to reflect a structural abnormality. The correlation between positive scans, simple partial seizures, focal neurologic signs and a focus on EEG were taken into account. If two or more features were present structural disease was detected in approximately half of the patients. Conversely when focal features were absent, the CT scan was abnormal in only 6% of the patients. Of the 52 patients with CT

abnormalities only 15 had lesions that were potentially treated with surgery and all these patients had one or more focal features. Thus fewer than 10% of the patients had their management influenced by CT.

Radioactive Isotopes

Such as technetium (Brain scan) are occasionally used for visualization of tumours, inflammatory masses, viral encephalitis, and some other vascular lesions. It is a simple non invasive but costlier procedure.

Magnetic Resonance Imaging (MRI)

Its use had permitted the visualization of cerebral lesions not evident on CT scan. The technique permits delineation of tissues without administration of contrast enhancing agents and because bone elicits no interference, it is particularly useful for visualising structures at the brain - bone interface i.e. in the posterior cranial fossa.

Positron Emission Tomography (PET)

It is an experimental investigative technique available only in few centres in the world (not available in India). Although these studies show great promise in the biochemical analysis of brain functions, the cost of the instrumentation and the technology required to produce isotopes will restrict PET scanning to major medical centres.

MATERIAL AND METHODS

MATERIAL AND METHODS

SAMPLE

The patients suffering from epilepsy who attended the out patient departments of Medicine, Paediatrics, Neurology and Psychiatry of Maharani Laxmi Bai Medical College, Hospital, Jhansi from July' 1987 to June'1989 formed the sample of the study. More than half of the patients were admitted for investigations. Patients of both sexes and every age group were included in the study. All patients had more than one seizure.

METHODS

All the patients were evaluated on the following lines :

1. History : detailed history of epilepsy.
2. Examinations :
   a. General examination.
   b. Systemic examination - especially of central nervous system.
   c. Fundus examination.
3. Investigations :
   a. Blood - sugar, urea.
   b. Serum - Calcium, VDRL.
   c. Cerebrospinal fluid - cytobiochemical and if necessary for malignant cells.

d. Radiological.

i. X-ray chest P.A. view.

ii. X-ray skull - AP view and lateral view.

iii. Computerised axial tomography, if possible.

e. Electro-encephalography - in all the cases.

f. Other relevant investigations.

History of epilepsy was obtained from the patients and from their relatives, after gaining the full confidence of patients and relatives. Special emphasis was given to the age of onset of epilepsy, frequency of fits, duration of illness, about the last attack, psychiatric problem, aetiological factors, family history of epilepsy, migraine, details of predisposing factors, premonitory symptoms, preictal, ictal and post ictal events were recorded. Patients were interrogated about intake of anti-epileptic drugs.

A general examination and a neurological examination was done in devery case. Relevant examinations of other systems were also done.

Electroencephalographic examinations were done in every case using an eight channel EEG recording machine. Ten-twenty system of electrode application was used. Both Monopolar (A, B, C and D run) and bipolar (E and F run) montage were taken in use. Routine recording and provocative procedures like - sleep recording, hyperventilation

and photic stimulation were done in all the cases. At times EEG was repeated, if required. Electroencephalographic diagnostic criteria were same as given by Kiloh et al (1982).

Other investigations were also done in every admitted case. CT scan were got done whenever possible.

At last the data were tabulated and analysed.

OBSERVATIONS

OBSERVATIONS

The present study was carried out on 200 patients of various types of epilepsies attending medical/neurological, psychiatric and pediatric out patient departments of M.L.B. Medical College, Hospital Jhansi from July, 1988 to May, 1989.

TYPES OF SEIZURES

The patients of primary generalised seizures (124 cases) dominated the study. The next common type of seizure in order of frequency were secondary generalised after focal onset (22%), simple partial (8.5%) and complex partial (7.5%).

TABLE 1 : Showing the types of seizures.

Types of seizures	No. of cases	Percentage
A. Primary Generalised	124	62.00
- Tonic-clonic - 120		
- Tonic - 1		
- Atonic - 1		
- Myoclonic - 2		
B. Secondary generalised with focal onset.	44	22.00
C. Simple partial	17	8.50
D. Complex partial	15	7.50
Total	200	100.00

FEATURES OF PRIMARY GENERALIZED EPILEPSY

TABLE 2 : Showing predisposing and aetiological factors in 30(30.6%) cases of primary generalised epilepsy.

Aetiological factors	No.of cases	Perce-ntage
Birth anoxia	6	4.08
Head injury	20	16.12
Inflammatory brain disease	4	3.28
Intracranial space occupying lesion	2	1.64
Fever	2	1.64
Migraine	2	1.64
Rheumatic heart disease	1	0.82
Tuberous sclerosis	1	0.82
Total	38	30.64

Three fourths of the patients in this study were having primary generalised epilepsy. Age of patients ranged from 1½ month to 60 years. There were 42% patients below the age of 10 years and 58% cases were above the age of 10 years in this study. Male and female ratio was 3:1. Frequency of the seizure varied from an attack in a year to several attacks in a day. About 46% patients were having seizures several times in a day, week or month. Five patients were admitted in status epilepticus. In 38(30.6%) cases, some predisposing factors could be worked out. Head injury was the commonest cause in 20 cases. Most common

cause was birth anoxia in 6(4.8%) cases. Other factors were inflammatory brain disease, intracranial space occupying lesion, fever, migraine, rheumatic heart disease and tuberous sclerosis (Table 2).

Precipitating factors were found in 18(14.5%) cases. In these cases most of the time seizures were precipitated by these precipitating factors but not always. Commonest factors was sleep - 12 cases(9.6%) sleep deprivation - 2(1.6%) cases, stress, emotion 2 (1.6%) cases and exertion-2(1.6%) cases.

TABLE 3 : Showing preconvulsive and post convulsive symptoms of primary generalised epilepsy.

Symptoms		No. of cases	Percentage
PRECONVULSIVE		7	5.60
Headache	- 1		
Vertigo	- 1		
Disinterest in environment	- 1		
An abnormal feeling	- 4		
POST CONVULSIVE		62	50.00
Headache & sleep both	28(40.3%)		
Sleep	13(20.9%)		
Headache	10(16.1%)		
Weakness	6(9.6%)		
Chakrabat	3(4.8%)		
Vomiting	2(3.2%)		
Drowsiness	1(1.6%)		
Running here & there	1(1.6%)		
Palpitation	1(1.6%)		

Preconvulsive symptoms were headache, vertigo, disinterest in environment and an abnormal feeling(Table 3). Post convulsive symptoms were found in 62(50%) cases. Among these 62 patients majority 25(40.3%) cases of them have these symptoms in the form of headache and sleep both. Next common symptom is sleep in 13(20.9%) cases and headache in 10(16.1%) cases, weakness in 6(9.6%) cases, ghabrahat in 3(4.8%) cases, nausea, drowsiness, running here and there, sweating and palpitation, all these were found in one case each(Table 3).

On doing neurological examination, 6 cases were mentally retarded, 3 cases were having behavioural abnormality, schizophrenia was found in 2 cases and one had third nerve palsy. This case of third nerve palsy had tuberculoma on CT scan and was presented as generalised seizures.

EEG FINDINGS

Out of 124 cases EEG was found abnormal in 89 cases. These abnormalities were seen in the form of generalised discharge, hypsarrhythmia, generalised brain damage, spyclonia and Lennox-Gastaut syndrome.

TABLE 4 : Showing EEG findings in clinical generalised seizures.

EEG findings	No.of cases	Percentage
Normal	68	52.40
Generalised	53	42.70
Hypsarrhythmia	1	0.80
Generalised brain damage	3	2.40
Myoclonic	1	0.80
Lennox Gastaut Syndrome	1	0.80
Total	126	100.00

CT scan was done in 17 cases out of which one had parietal lobe granuloma. One had secondary metastatic deposits and one had calcified patch in frontal area.

TABLE 5 : Showing the findings of CT scan.

Findings	No. of cases
Normal	14
Parietal lobe granuloma	1
Secondary metastatic deposits	1
Calcified patch in frontal area	1
Total	17

Most investigations like VDRL, blood sugar, serum calcium and X-ray skull showed no abnormality in any of the case, but ideally all these investigations should be done in all the cases.

FEATURES OF SECONDARY GENERALIZED EPILEPSY

There were 44 (22%) cases of secondary generalised epilepsy in this study. Age varied from 2 months to 30 years. Twenty cases were below the age of 10 years and remaining 24 cases were above 10 years of age. Frequency of seizures varied from several attacks in a day to few attacks in 6 months. Majority of the cases (18) cases had seizures several times in a month.

Out of 44 cases, 11(25%) cases had aetiological factors in the form of head injury in 6 cases, inflammatory brain disease in 2 cases and intracranial space occupying lesion in 1 case.

Precipitating factors were found in 8 cases in the form of sleep(4 cases), fatigue (1 case) awakening (2 cases) and seeing cinema (1 case). Premonitory symptoms were present in 2 cases in the form of headache and dizziness.

Among 44 cases clinical evidence of focal onset was evident in 15 cases in the form of motor features - 13 cases, sensory features in 2 cases, and autonomic features in 2 cases while 27 cases had clinically generalised features with only EEG evidence of focal onset.

Post convulsive features were found in 9 cases in the form of headache-3 cases, weakness 2 cases, sleep-1 case, vomiting-1 case and todds sleep-2 cases.

TABLE 6 : Showing clinical features and post convulsive features of secondary generalised epilepsy.

Clinical features	No.of cases
Autonomic	2
Focal	15
Motor - 13	
Sensory - 2	
Generalised	27
Post convulsive	9
Headache - 3	
Todds palsy - 2	
Sleep - 1	
Vomiting - 1	
Weakness - 1	

Associated neurological features were found in 3 cases (Table 7).

TABLE 7 : Showing the neurological features.

Features	No.of cases
Mental retardation	1
Hydrocephalus	1
Papilloedema, tuberous meningitis and third nerve palsy.	1
Total	3

EEG FEATURES

Out of 44 cases, 12 cases had normal EEG, 3 cases showed generalised epileptic discharge. 12 cases showed focal and 17 cases showed focal with secondary generalisation (Table 8).

TABLE 8 : Showing EEG features of secondary generalised epilepsy.

EEG features		No.of cases.
Normal		12
Focal getting secondary generalised		17
Focal		12
	Bilateral - 2	
	Left - 4	
	Right - 2	
	Central - 4	
Generalised		3
Total		44

CT scan could be performed in 5 cases out of which one showed tuberculoma. Rest of the 4 CT scan were normal.

SIMPLE PARTIAL SEIZURES

Among 200 cases, 17(8.5%) cases had simple partial seizures. Their age ranged from 8 to 40 years. Seizure frequency varied from several attacks per day upto one attack per year. Several attacks in a week was observed in 5 cases. Predisposing factors was seen in 5 cases(19%). Out of which 3(17.6%) cases had head injury and 2(11.7%) cases had birth injury. One patient had monoplegia. No other clinical features was observed.

TABLE 9 : Showing clinical features of simple partial seizures.

Clinical features		No.of cases	Perce-ntage
Convulsive			
Right side of body		8	47.05
: Motor - 8			
Left side of body		9	52.94
: Motor - 7			
: Sensory-2			
Post convulsive		7	41.17
Todd's palsy	- 2		
Headache	- 3		
Sleep	- 1		
Generalised weakness	- 1		

Out of 17 cases, 14 had clonic movements and 3 had sensory symptoms in the form of tingling numbness and burning in feets. In 9 cases seizure started from finger and thumb. Out of 17 cases, 8 had right sided symptoms and 9 had left sided symptoms.

Post convulsive features were found in 7 cases, out of which 2 had todd's palsy, 3 had headache, and sleep and generalised weakness was observed in one case.each.

CT scan could be performed in 4 cases, out of which one case showed intracerebral haemorrhage. Rest of the 3 cases had normal CT scan.

EEG FINDINGS

Out of 17 cases, 8 cases had shown a normal EEG, while 9 cases had shown focal features in the form of phase reversals, sharp waves and slowing.

TABLE 10 : Showing EEG features in simple partial seizures.

EEG findings	No.of cases
Normal	8
Focal	9
: Right hemisphere - 3	
: Left hemisphere - 1	
: Central - 5	
Total	17

COMPLEX PARTIAL SEIZURES

Out of 200 cases 15 cases were of complex partial seizures. All the patients were below the age of 30 years. Male : female ratio was 4:1. Frequency of seizures varied from several fits in a day to one

fit in a year. Majority of the patients (9 cases,60%) were having several fits within a week. The only predisposing factor was in the form of head injury in 2(13.3%) cases. The details of clinical features are shown in table ii.

TABLE ii : Showing clinical features of complex partial seizures.

Clinical features		No.of cases	Percentage
Aura		9	60.00
: Visual hallucination	- 3		
: Auditory hallucination	- 1		
: Somatosensory hallucination	- 1		
: Fear	- 1		
: Sexual excitement/desire	- 1		
: Dizziness	- 2		
Automatism/motor phenomena		9	60.00
: Walking in a circle	- 1		
: Wandering	- 1		
: Lateral deviation of head and eye	- 1		
: Hypotonia	- 1		
: Smacking of lips	- 2		
: Running	- 1		
: Looking up	- 2		

Blood and CSF examinations were normal. Only 3 patients could afford CT scan out of which one showed granuloma in temporal lobe.

EEG FINDINGS

Table 12 shows that the EEG findings were positive in 10(66.67%) cases out of 15 cases. EEG was normal in rest of the patients.

TABLE 12 : Showing EEG findings in complex partial seizures.

EEG findings	No.of cases	Percentage
Normal	5	33.33
Temporal lobe focus	10	66.67
: Right - 5		
: Left - 4		
: Bilateral - 1		
Total	15	100.00

D I S C U S S I O N

DISCUSSION

Epilepsy is a common illness. Five per thousand population seems to be affected (College of General Practitioners, 1960). The overall prevalence rate per 1000 of the general population vary from 1.3 in Formosa (Lin, 1953) and 1.5 in Niigata, Japan(Sato, 1964) to 9.2 in Warsaw (Zeilinski, 1974a) and as high as 19.5 in Bogota, Colombia (Gomez, Arvinieges and Torres, 1978). In over half of the studies the rates lie between 4 and 10 per thousand. In most of the studies where rate was lower than 4 per thousand information was obtained solely from a review of medical records or from general practitioners. Considering prevalence rates of 9/1000 (Mathai, 1971) and accepting this figure for the whole country millions of epileptics are acceptable. The workload of investigating these patients is a difficult task especially in India where neurological centres with specialised facilities are few. Most of the cases of epilepsy remained undiagnosed for long time as the disease is still not accepted as organic illness by a large number of people in our society. Disease is thought to be caused by devil so many people seeks faith in healing by itself. There has been considerable progress in the epileptology over the past two to three decades.

With the help of the sophisticated EEG related techniques, the diagnostic part of the disease is improved. Considering the problem of epilepsy and new developments especially in diagnostic field we had concluded a clinical and EEG study of epilepsy.

GENERAL DATA ANALYSIS

AGE

Age specific prevalence rates in several studies (Brunis et al, 1966; Juul-Jensen, 1974 and Maurer et al, 1986) have been found to be lowest in the first decade and rising gradually upto the fourth decade. In this study majority of the cases had first attack at second decade, then there was a gradual decline, while in other studies, majority of the cases had first attack in first decade.

SEX

In this study slightly more than one fourth of the patients were females. This finding is exactly similar to the study done by Dixit et al (1989). Females were once said to suffer from epilepsy slightly more frequently than males. In Gowers study of 3000 cases the ratio of female to male was 11:12. However the sex incidence is in changing patterns. In one study male/ female ratio was 1.5 : 1(Zielinski, 1977). Similar observations were found by Virmani and Sarmay, 1964; Reddy, 1971 and Agnihotri et al, 1972.

In the present study male dominance might be because of Indian society especially in rural population gives more importance to males because they are the earning members of the family.

CLINICAL FEATURES

It is important to know the prevalence of various seizure types in epileptic population. There had been many studies on clinical classification. In the present study, the seizures have been classified on the basis of the classification given by Marsden and Reynold (1982). Seizures were classified as generalised, partial secondary generalised and unclassifiable. Gastaut et al (1975) studied 6000 epileptics and they worked out their different classifiable groups and their relative frequency in children and adults. A similar study (Joshi et al. 1977) has been carried out in India. Both these studies were based on classification almost similar to that used in the present study. Primary generalised seizures dominated our series (62%) in comparison to 38% in the series of Gastaut et al (1975), 28% in the series of Joshi et al, 1977 and 51.7% in the study of Dixit et al, 1980. In the above studies of Gastaut et al, 1975; Joshi et al, 1977 and Dixit, S.S., 1980, the number of cases of absence seizure, was 10%, 3% and 0% respectively. We had come across a case of Lennox Gastaut syndrome, which is a petit mal variant. This could be because of

a smaller sample of the study. Cases of secondarily generalised was frequently found in our study (22%) as compared to the above study of Gastaut et al (1975)(12%) and Joshi et al, 1977 (13%). The study conducted last year in this institution by Dixit S.S. (1989) has shown 24.2% cases of secondary generalised which is approximately similar to this study. In our study the percentage of simple partial seizure was low in comparison to those observed by Joshi et al, 1977 (32%). On the other hand frequency of complex partial epilepsy was almost equal (6.5%) in this study and(7%) in that of Joshi et al,1977.

FEATURES OF PRIMARY GENERALISED SEIZURES

This was the commonest type of seizure. The patients included in this study having generalised features and unconsciousness without any focal onset or presence of aura. It included idiopathic and symptomatic epilepsy both. In many patients frequency of seizure was high. 40% of cases had seizures several times a day, biweekly, weekly or monthly. Earlier to this maximum frequency of seizure was reported by Sharma (1987) where one third of the cases had seizures several times in a month. Figure of our study is higher than the figure in the study conducted by Sharma. This is possibly because in our community people seeks medical advice when the illness gets advanced and disabling.

Out of 124 cases of primary generalised seizures some aetiological factors was found in 38(30.6%) cases. In the study of Sherwon (1987) same cause or other could be detected in about one third of the cases of all epileptics. The figure more or less nearer to the present. study. In the present study aetiological factors were in the form of head injury in 20(16.12%) cases, birth anoxia in 6(4.8%) cases, inflammatory brain disease in 4(3.2%) cases. Rest of them were intracranial space occupying lesion, fever, migraine, rheumatic heart disease and tuberous sclerosis. We had 14(11.29%) cases who was the eldest child of the family. This is possibly because of increased liability of the first born to head injury during birth. In the study of Kaarer et al (1986) it was shown that the head injury (26.9%) was the commonest cause of epilepsy and the least common cause was the birth injury (3.3%) while intracranial infections caused epilepsy in 18.9% cases.

Our figures in respect to head injury are less than the above mentioned study. This may be because of the majority of the cases of primary generalised epilepsy having head injury were in the paediatric age group. Children often conceal head injury from their parents; secondly healing of wound is better in childhood. Most of the children do not remember about the injuries which use to occur during playing. As compared to the study of Kaarer et al (1986), the incidence of birth injury was

slightly higher, possibly because of lack of sophisticated obstetric services especially in rural population of India.

In this study migraine, rheumatic heart disease and tuberous sclerosis were also seen as an aetiological factor. Though loss of consciousness at the height of headache is usually syncopal there is slightly increased incidence of epilepsy in migraine sufferers, even in those who have no evidence of cerebral lesion. A possible role of tyramine in the physiological mechanism of the two disorders was postulated by Scott, Moffett and Swash (1972). In present study, 1 case was of rheumatic heart disease having mitral stenosis along with mitral regurgitation but no evidence of embolism was seen. Epilepsy associated with rheumatic heart disease more often than can be explained by chance. In mitral valve disease small cerebral emboli may cause epilepsy as may a small asymptomatic infant due to intracranial atheroma (Dodge, Richardson and Victor, 1964). In this present study one case had tuberous sclerosis having adenoma sebaceum convulsions, shagreen patch and normal intelligence and EEG evidence of generalised seizures. Tuberous sclerosis (Epiloia) is manifested by a clinical triad of convulsive seizure, mental deficiency and adenoma sebaceum. Pathological discription of tuberous sclerosis is given by Urich (1976). Our patients had convulsive

seizures and adenoma sebaceum but the intelligence was normal. This is possible because now some partial forms of tuberous sclerosis is increasingly recognised. The three cases reported in detail by Davidson and Vinson (1961) were of superior intelligence. Although all the cases had radiological evidence of cerebral involvement as well as lesion of tuberous sclerosis elsewhere. Lagos and Gamez (1967) reviewed the records of 71 patients with tuberous sclerosis studied at the mye clinic, 26 of the 66 patients on whom there were records of intellectual capacity had normal intelligence, 18 of these 26 had seizures.

Preconvulsive features were present in 7(5.6%) cases. They were in the form of headache, vertigo, disinterest in environment and an abnormal feeling.

Post convulsive features were seen in 62(50%) cases in the form of headache in 10(16.1%) cases, sleep in 13(20.9%) cases, headache and sleep in 25(40.3%) cases, weakness in 6(9.6%) cases and ghabrahat in 3(4.8%) cases. Rest of the features were nausea, drowsiness, running here and there, sweating and palpitation. These features were found more in adults. According to Laidlaw and Richens (1962) children to not go through post-ictal phase and if these are present then they usually recovers completely within a minutes.

On doing neurological examination, 6 cases were mentally retarded, 3 cases had behavioural abnormality, 2 cases had schizophrenia and one case had third nerve palsy.

In present study 6(4.8%) cases were of mental retardation. In the series of 3600 cases of all types of epilepsy including 2000 of generalised epilepsy. Sharma (1967) found neurological deficit in 9.8% cases, mental retardation in 40% and behavioural abnormalities in 50% cases. There were highest number of cases of mental abnormality in the present study which is probably due to birth anoxia because of most of the deliveries were conducted in the rural area and most of the time history of birth injury or anoxia was not illicitible. In a study of Brown et al (1976) it has been shown that out of 94 patients having birth anoxia 32 cases had neurological deficit and 46 had convulsions.

No other investigations except ECG, CT scan and CSF examination showed abnormality. VDRL, blood sugar, serum calcium and X-ray skull showed no abnormality.

CT scan could be done only in 17 cases out of which 14 of these cases were normal. 1 had parietal lobe granuloma and 1 had cerebello pontine angle tumour, 1 patient had secondary metastatic deposit and 1 had calcified patch in frontal area.

In CT scan, other studies were conducted by Gastaut (1976) who observed 34.51% abnormal cases in series of 1782 cases. Young et al (1979) scanned 256 children out of which 33% abnormalities were seen. In the present study 1 patient had only 2 seizures at a one month gap, whose CT scan showed a granuloma. Young et al (1982) showed that even a solitary focal seizure is likely to result a structural abnormality.

EEG FINDINGS AND CLINICAL CORRELATION

EEG abnormalities were present in 99(47.59%) cases of generalised epilepsy. This figure is higher as compared to other studies i.e. 30-40% (Kiloh et al,1982). This could be explained on account of higher number of children, higher frequency of seizures and many EEG were done within twenty four hours of the last attack. Out of 18 patients having seizures several times a day EEG findings were normal only in 3 patients. EEG findings were generalised in 53 patients, hypsarrhythmia, myoclonic and Lennox Gastaut syndrome in 1 patient each.

We had some nurses , 5 patients of mental abnormality out of which 3 patients had shown generalised brain damage in the EEG. Generalised convulsions were a common feature of brain damage.

One EEG showed hypsarrhythmia. Patient was 5 years old, who had encephalitis one year back, had generalised convulsions. He was mentally abnormal. His

EEG showed spike and polyspike discharges on continuously abnormal background of diffuse high voltage arrhythmic slow activity. In a series of children Friedman and Pampiglione (1971) showed hypsarrhythmic EEGs during the first year of life. There was a mortality of 26% cases. The majority of them died before the age of 3 years. One case of myoclonic epilepsy has shown the typical features of myoclonic epilepsy. EEG showed high voltage spikes and slow waves which were bilaterally synchronous. The spikes were frequently multiple occurring in groups of two to six. This finding was similar to that of Gastaut (1954b) who called these complexes as 'polyspikes and waves'. One case showed the EEG findings of Lennox-Gastaut syndrome, clinically having tonic seizures and most of the time the frequency of spike and wave pattern was two and a half Hz. This finding was more or less similar to the finding of Aicardi (1982). who described the syndrome clinically by brief atonic, myoclonic and tonic seizures and electrically by atleast some 2 Hz spike wave activity. The frequency may even be faster (3 Hz) spike wave paroxysms. Family history was negative.

FEATURES OF SECONDARY GENERALISED EPILEPSY

In the present study out of 200 cases, 44(22%) cases were of this type of epilepsy. In this study clinically 17 cases were found to have focal features with secondary generalisation, another 27 cases who were

clinically thought to be the cases of primary generalized seizures, were found to have the evidence of focal onset in EEG. This might be possible because of rapid spread of focal discharge.

Majority of the cases(18, 40.9%) had seizures several times in a month. This figure is similar to that of generalized epilepsy.

Aetiological factors were present in 11(25%) cases in the form of head injury - 8 cases, inflammatory brain disease - 2 cases, intracranial space occupying lesion - 1 case. This figure is slightly less than that of primary generalized seizure (28.5%). In a series by Joshi et al (1977) aetiological factors were present in 60% cases. The lesser incidence of aetiological factors was because we could perform CT scan only in 5 cases, which is now the essential investigation in the seizures of focal onset. According to Gastaut and Gastaut (1976) CT scan detects 20% more cerebral lesions than the combination of long established techniques like skull X-ray, EEG and angiography.

Precipitating factors were found in 8 cases (18.1%) in the form of sleep - 4 cases, fatigue - 1 case, awakening - 2 cases and seeing cinema - 1 case.

Premonitory symptoms were present in 2 cases in the form of headache and dizziness.

CLINICAL SEIZURE PATTERN

Out of 44 cases, 15 cases had focal features in the form of tonic spasms, twitching at the angle of the mouth or in the fingers or clonic movements in the one upper limb and in the lower limb on the same side. Two cases had tingling numbness in lower limbs and one case had abnormal sensation in lower extremities. Autonomic features were present in two cases in the form of epigastric sensation. 27 cases had generalised convulsions.

Associated neurological features were found in 3 cases. These were having mental retardation - 1 case, schizophrenia - 1 case and tubercular meningitis with papilloedema and third nerve palsy in one case. EEG of this patient showed focal epilepsy of left side with secondary generalisation. CT scan had shown parietal lobe granuloma in left side. Clinically he was having focal onset on the right side of the limbs.

EEG FINDINGS AND CLINICAL CORRELATION

In 44 cases, 12 cases had an normal EEG. 17 cases had shown a focus getting secondary generalised and 12 cases had shown a focus in EEG but clinically generalisation was seen.

There were 3 such cases who have generalised discharge in EEG but clinically only focal features were seen. Among these cases 4 cases had a central focus.

In 2 cases bilateral focus was seen. This is probably because of mirror image (Kiloh et al, 1962) due to commissural fibres in the brain.

FEATURES OF SIMPLE PARTIAL SEIZURES

In our study out of 200 cases 17(8.5%) cases were of simple partial seizures. The percentage was more or less similar to that of complex partial seizures. Percentage of this series is more (16.8%) than the study conducted last year in this institution. Other studies reported 20% simple partial seizure (Sherven (1987) whereas 58% and 62% cases of simple partial seizure were reported by Gastaut et al (1975) and Joshi et al (1977) respectively. Age ranged from 8-40 years. None of them had status epilepticus. In this series also frequency of seizures was high. Out of 17 cases 5 had seizures several times in a week. Predisposing factors were found in 5(29%) cases out of which 3(17.6%) cases had head injury and 2(11.7%) cases had birth anoxia. All the 3 cases of head injury were 2-5 months old. Joshi et al (1977) had found head injury in 13% cases. They found some aetiological factors in 30% cases. Neurological deficit was found in one case in the form of monoplegia. A lady of 22 years of age had focal clonic movements in her left leg after which she had monoplegia in the same leg associated with vomiting. Her CT scan

revealed large intracerebral haemorrhage. She died after two days of the epileptic fit. In the simple partial seizure associated neurological deficit was found only in one case as compared to focal with secondary generalisation where neurological deficit was seen in 3 cases.

CLINICAL SEIZURE PATTERN

Out of 17 cases, 14 had focal clonic movements and 3 had sensory symptoms in the form of tingling numbness and burning in feets. In all the 9 cases seizure started from finger or thumb except in one case. Out of 17 cases 8 had right sided symptoms and 9 had left sided symptoms. Start of seizure from thumb or finger may be due to representation of hand in brain is under prominent part of skull and is more prone to injuries as compared to toes and angle of the mouth. Post convulsive features were found in 7 cases in the form of weakness, todd's palsy, headache and sleep.

EEG FINDINGS IN SIMPLE PARTIAL SEIZURE

Out of 17 cases, EEG abnormalities were found in 9(53%) cases. Out of these 9 cases, all the cases showed focal abnormality. Out of which 3 cases had right sided focus. One case had left sided focus and 5 cases had a central focus.

In above findings 2 cases were having right sided focus and convulsions were occurring in right side. No explanation could be given to this finding. The same finding was observed by Dixit, S.S. (1989).

CT scan could be performed in 4 cases. One showed large intracerebral haemorrhage. EEG of this patient was normal. In study by Yang et al (1983) approximately half of the patients with simple partial seizures had positive scan. In this study only one CT scan had finding. This may be because we were not able to perform CT scan easily because of its cost factor and non availability in this city.

FEATURES OF COMPLEX PARTIAL SEIZURES

Psychomotor epilepsy is a clinical finding while temporal lobe epilepsy is a EEG finding. Not all the cases of psychomotor epilepsy have a temporal lobe focus and not all the patients with temporal lobe focus in EEG exhibit psychomotor seizures(Stevens, 1966). Out of 200 cases, 15(7.5%) cases turned out to be the case of complex partial seizures.

All the patients were below 30 years of age. Seven were females and ten were males.

Shukla (1975) found that majority of the cases had the onset of seizures below the age of 20 years. Most of them had their first fit before the age of 10 years. This observation was against the earlier belief

that temporal lobe epilepsy was a late form of convulsive disorder. Gibbs et al (1943) found that only 9.3% cases of epilepties below the age of 20 had temporal lobe epilepsy. Stevens (1966) found that temporal lobe epilepsy was the disease of adults. Currie et al (1971) reported maximum incidence of onset of seizure in third and fourth decade of life in their study of 666 cases of emporal lobe epilepsy. However, Aird et al (1967) reported that 42% cases had their first attack in the first decade and over 78% in the first two decades of life. Virmani and Sachdev (1966) and Reddy (1971) drawn the similar conclusions. This apparent contradiction may be explained by improvement in the methods of diagnosis and by the fact that in children the initial attacks are not of the classical psychomotor type and are easily missed (Falconer and Taylor, 1970). Most of the patients had onset of seizures from 2 months to 2 years. In the series of Reddy (1971) mean age was 6.42 years. Agnihotri et al (1972) in a series of epilepsy in general, found the mean duration to be 7.5 years. In both these studies patients were either taking irregular treatment or no treatment was taken at all. In this present study out of 15 patients, 5 were taking regular treatment, 7 cases started taking treatment after being fully investigated and none were taking treatment regularly. Explanation for regular treatment in these cases is because the illness in these patients was distressing

and making them unable to perform their professional work. Out of which most distressing was transient loss of memory in 4 cases, and in 6 cases the frequency of seizures was high i.e. daily or biweekly. In the series of Shukla et al (1979) about two thirds of patients had seizures daily or weekly which is more or less comparable with this series. Lesser frequency was reported by Currie et al (1971) where two thirds of the patients had fits twice a month. Out of 15 cases, only 2(13.3%) cases had an aetiological factors in the form of head injury. In the study conducted last year in this institution (Dixit, 1989) out of 9 cases only 1 case had aetiological factor in the form of cerebrovascular accident which he had few months prior to the onset of the seizures. This is more or less similar to our study. In the literature workers could not get aetiological factors in 22.9% -89% of cases of their series (Falconer et al, 1964; Aird et al, 1967; Reddy, 1971 and Mohan, 1974).

Lesser percentage of aetiological factors in this present study may be probably because of the smaller sample and secondly people were not able to go for CT scan (because it is an expensive investigation), which detects 30% more cerebral lesions than the combination of long established techniques (Skull X-ray, EEG and angiography etc.).

Two (13.3%) cases had a positive family history while family history was positive in 11.1% cases of Dixit (1989) and 14.2% cases of Shukla et al (1979).

CLINICAL SEIZURE PATTERN

Out of 15 cases, 13 had feature of psychomotor epilepsy and 2 cases had features of grand mal epilepsy but EEG had shown a focus in temporal lobes. Aura occurred in 9(60%) patients out of 15 cases. It was in the form of visual hallucinations in 3 cases, voices heard in the case-1, hyperasmality in 1, fear in 1, wandering in 1, somato sensory in 1 and dizziness in 1. Currie et al (1971) reported visual aura in 18%, auditory in 16% and olfactory in 12%. Aura in the form of laughing and crying can occur in the same patient (Often et al, 1971 and Sethi and Surya Rao, 1976).

Aura has been found in 60% of cases (Shukla et al, 1979). The commonest was visual hallucinations (as in this present study) followed by vertigo and epigastric sensation. Olfactory and gustatory auras once said to be diagnostic of temporal lobe seizures are in fact rare(De Jong, 1957 and Penfield, 1964). In our study out of 15 cases, behavioural abnormality was found in 1 case. A significant greater number of temporal lobe epileptics do have emotional disturbances in childhood and psychiatric abnormalities in later part of life, in comparison to patients with grand mal epilepsies (Shukla et al, 1979).

Out of 15 patients of temporal lobe epilepsy one patient became hypersexual during the attack. Abnormal sex behaviour associated with temporal lobe epilepsy has frequently been described in man (Gastaut and Calemb, 1954; Marchini and Sinisi, 1957 and Hierons and Saunders, 1966) and in animals (Kluver and Bucy, 1939). Occurrence of hypersexuality in association with temporal lobe epilepsy is rather rare(Taylor, 1966b). In this study of 100 patients only one was hypersexual. A large number of temporal lobe epileptics were found to be hypersexual (Shukla et al, 1979).

Motor phenomena was present in 2(13.3%) cases in the form of lateral deviation of head, eye and hypotonia. In study of Bansi (1964), 10% of psychomotor epileptics had motor symptoms. In the present study automatism was present in 7 patients in the form of smacking of lips, running, looking up, wandering and walking in a circle. Motor phenomena and automatism have been described under the same heading of automatism (Dichter, 1967). Combination of motor phenomena and automatism is a essential feature of complex partial seizures as majority of the cases had these features.

Out of 15 patients, 3 had generalised tonic clonic seizures and 2 had focal clonic seizures. Several authors include these sensorimotor manifestations as a clinical features of psychomotor seizures (Bansi et al,

1984). Lennox (1960) described psychomotor seizure as characterized by 'automatic' subjective and tonic focal signs.

EEG FINDINGS

The investigation which showed positive findings was the EEG in all cases and CT scan in 1 case.

EEG showed finding in 10(66.67%) cases out of 15 cases. In the present series 5 cases had normal EEG. Out of 10 abnormal EEG, all showed a temporal lobe focus. Among these 10 cases of temporal lobe focus 5 cases had very frequent discharges that too get enhanced in sleep recording. Five cases had right temporal focus and 4 had left temporal focus and 1 had bilateral temporal focus.

Only 4 patients could afford CT scan. Granuloma in temporal lobe was observed in one case. EEG of this case showed a mirror image.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present work entitled "A continued study on epilepsy - Clinical and Electroencephalographic Aspects" was carried out on 200 patients of various types of epilepsies. A detailed history was taken from the patients and as many relatives there of as possible. Detailed physical examination was done in every case, stressing more on the nervous system. Various laboratory investigations including ECG was done in every case. In some cases CT scan could be performed.

Age of the patients ranged from one and a half month to sixty eight years. The sample has primary generalised seizures 124 (62%), secondary generalised 44(22%), simple partial 17(8.5%) and complex partial 15 (7.5%).

Aetiological factors were found in 56 cases out of which in primary generalised 38(30.6%) cases, secondary generalised 11(25%) cases, simple partial 2(13.3%) cases. Among these 56 cases, majority(33 cases) of the patients had head injury. Next commonest aetiological factor was birth anoxia (8 cases). Rest of the cases had inflammatory brain disease, intracranial space occupying lesion, fever, migraine, rheumatic heart disease and tuberous sclerosis.

Precipitating factors were found in 26 cases out of which 18 cases were of primary generalised and 8 cases were of secondary generalised epilepsy. Commonest

precipitating factor was sleep (16 cases). Rest of the cases had precipitating factors in the form of sleep deprivation, awakening, seeing cinema, stress, emotion and exertion. Preconvulsive symptoms were found in 9 cases out of which 4 cases had preconvulsive symptoms in the form of abnormal feeling. Others had headache, dizziness, vertigo and disinterest in environment. Post convulsive features were found in 70 cases out of which 62 cases were of primary generalised epilepsy. Among 70 cases, 22 cases had headache and sleep both, 16 cases had headache and 15 cases had sleep. Rest of the cases had weakness, ghabrahat, drowsiness, sweating, palpitation and running here and there. Four cases had todd's palsy, among these cases 2 cases had simple partial epilepsy while 2 cases had focal with secondary generalisation. Clinical features other than these of epilepsy were present in 16 cases, out of which 7 cases had mental retardation. Rest of the cases had behavioural abnormality, schizophrenia, third nerve palsy and monoplegia.

Except EEG and CT scan almost all the investigations were normal. CT scan could be performed in 29 cases, out of which 6 CT scan had abnormality in the form of granuloma, secondary metastatic deposits, calcified patch and intracranial haemorrhage.

Out of 200 cases EEG was abnormal in 110(55%)

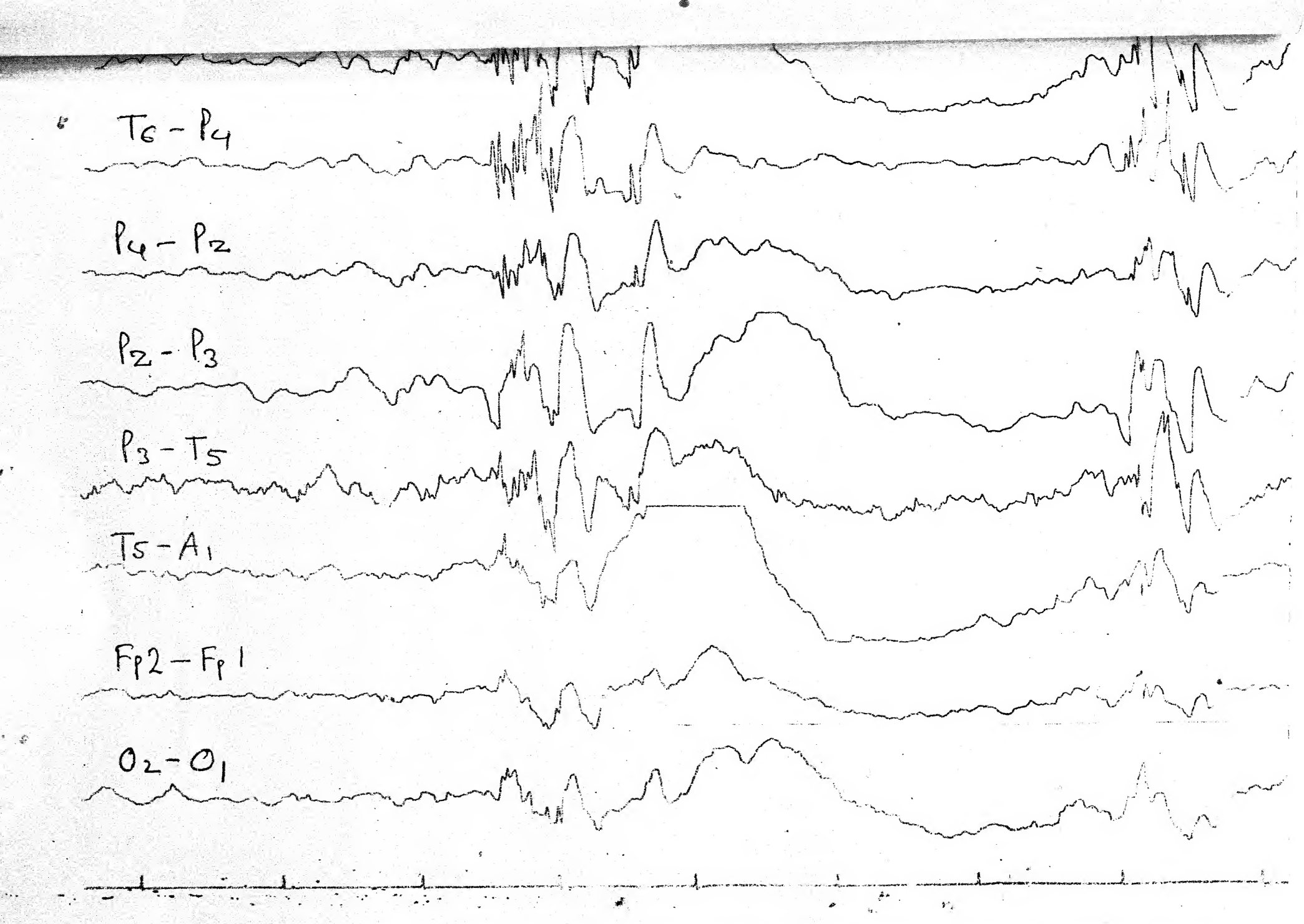
cases. In primary generalised 39 cases had abnormal EEG in the form of generalised epileptic discharge in 33 cases, generalised brain damage in 3 cases, hypsarrhythmia, lennox gastaut syndrome and myoclonic in one case each. In focal with secondary generalisation 32 cases showed abnormal EEG. In simple partial seizures 9 cases had positive EEG showing focal features while complex partial seizures had shown 10 cases having a temporal lobe focus.

EEG no. 240 : Showing spike and wave pattern of generalised seizures.

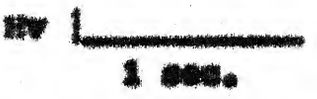

T6 - P4
P4 - Pz
P2 - P3
P3 - T5
T5 - A1
Fp2 - Fp1
O2 - O1

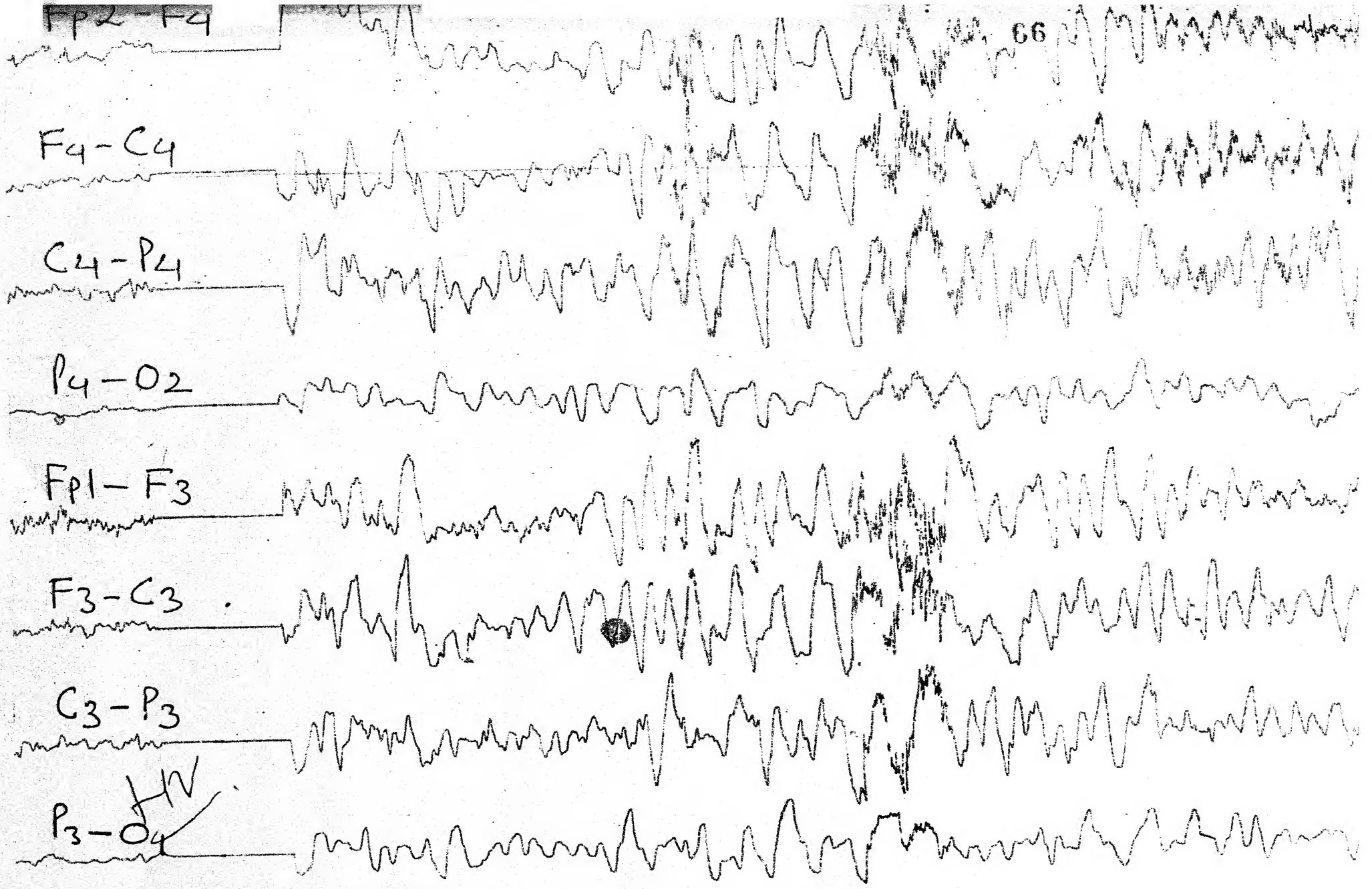
EEG no. 209 : Showing polyspikes:record showing generalised seizures.

50 mv |_____


T6 - P4
P4 - Pz
Pz - P3
P3 - T5
T5 - A1
Fp2 - Fp1
O2 - O1

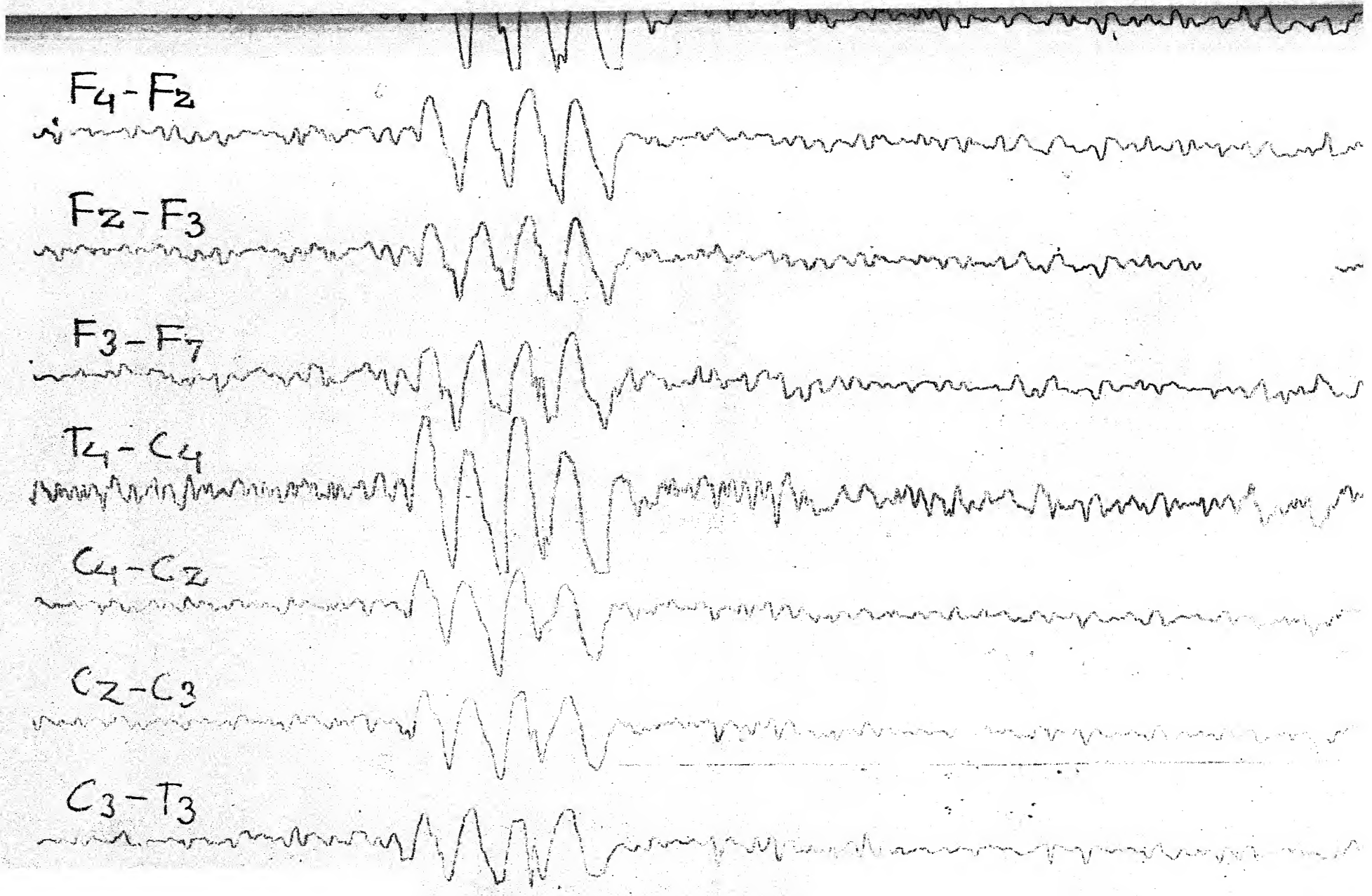
EEG no. 269 : Showing generalised slowing seen after hyperventilation.



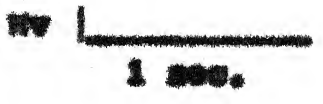


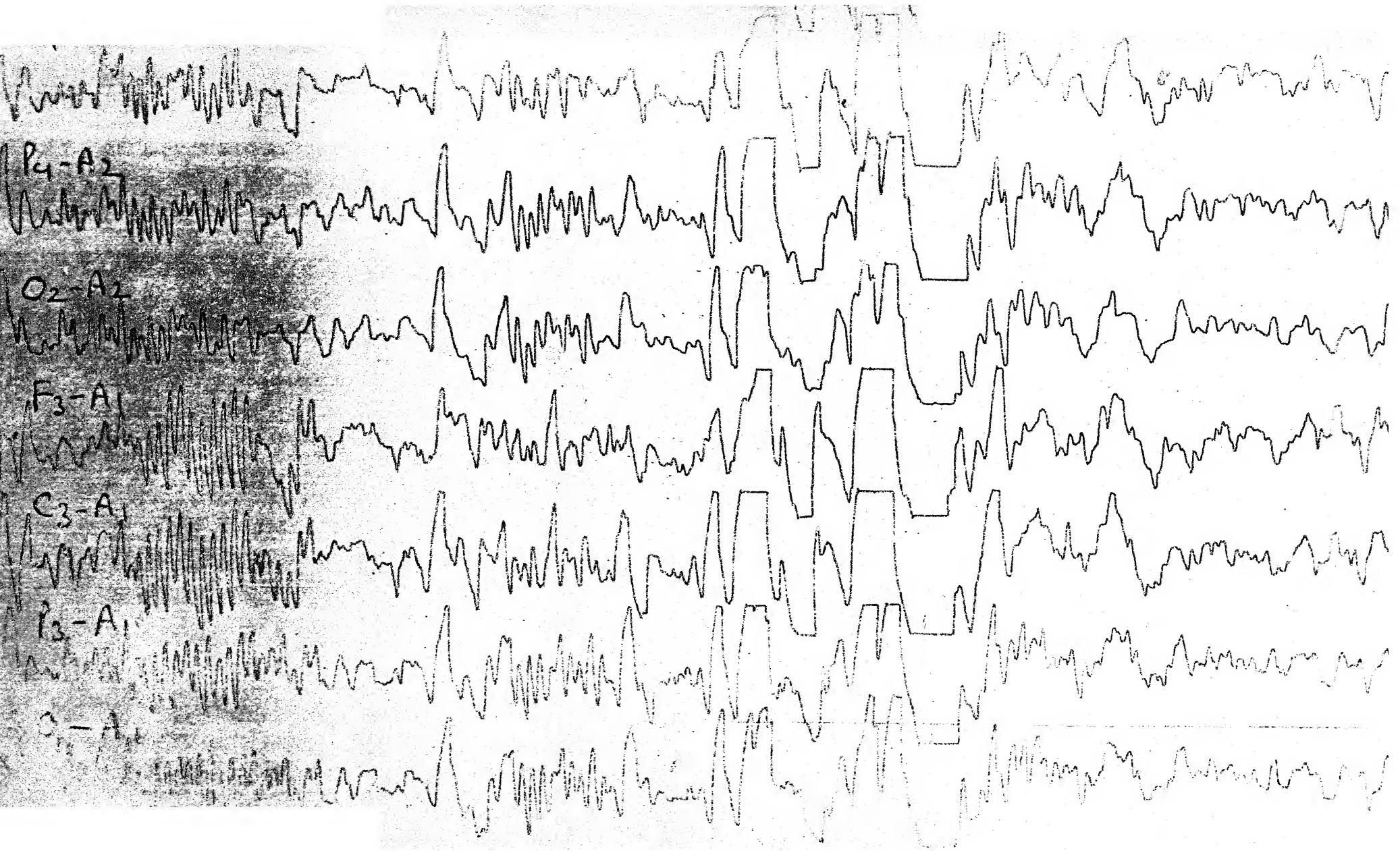
HYPERVENTILATION.

EEG No. 200 : Showing slowing indicative of generalized epilepsy.


F4-F2
F2-F3
F3-F7
T4-C4
C4-C2
C2-C3
C3-T3

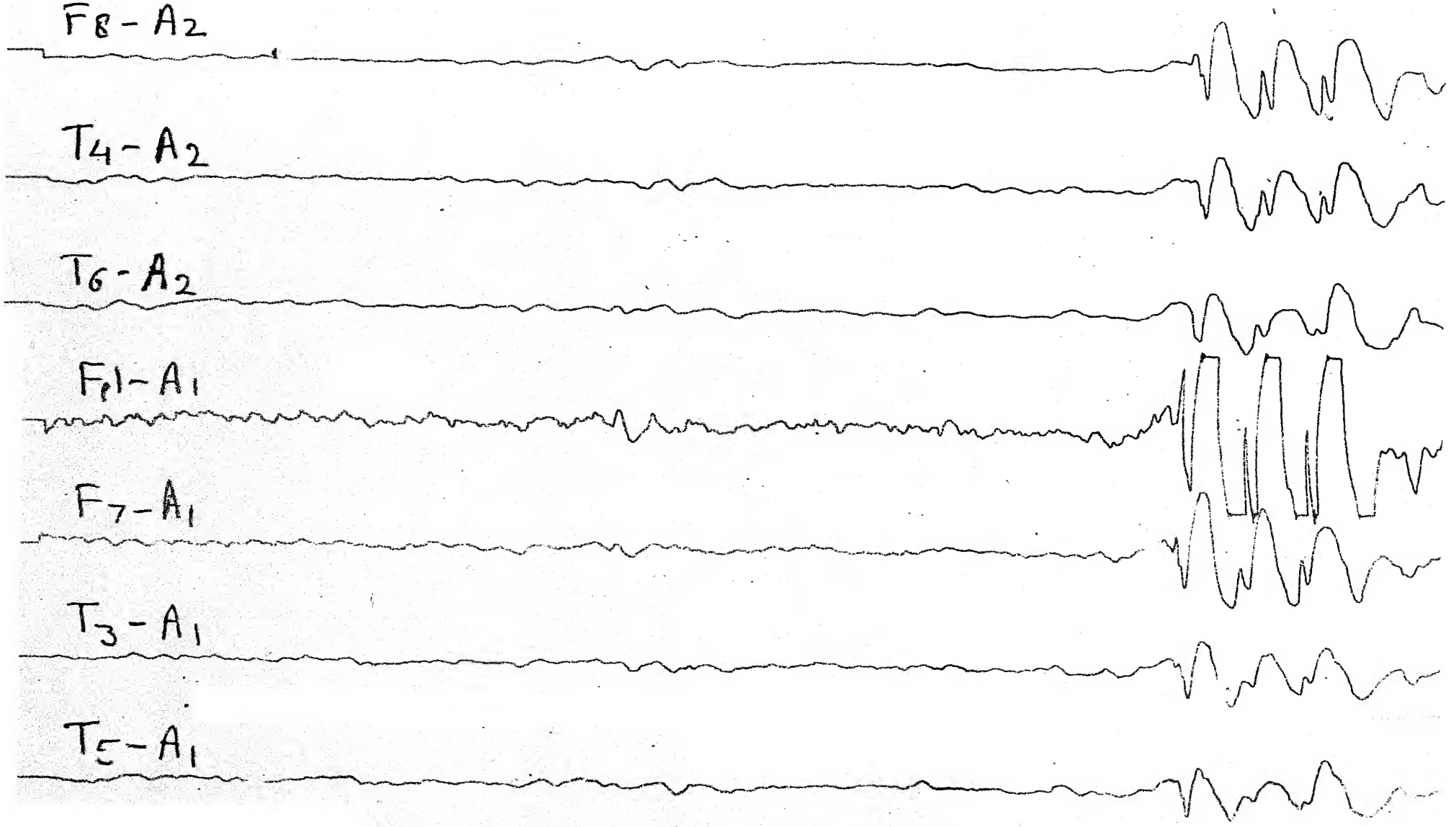
ETC No. 164 : Showing sleep spindles and generalized slowing.



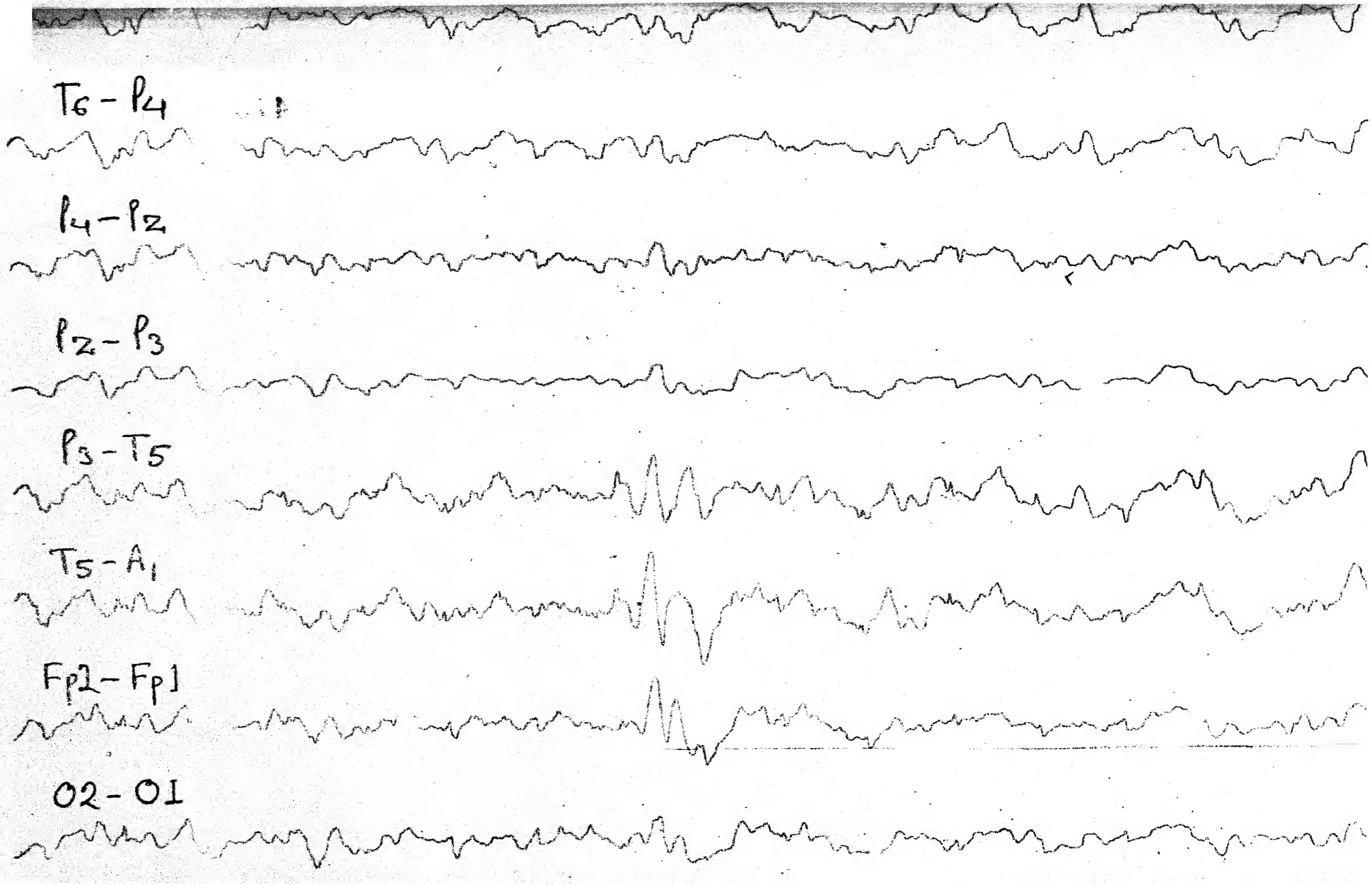

P4-A2
O2-A2
F3-A1
C3-A1
P3-A1

EEG No. 211 : Showing Lennox Gastaut syndrome

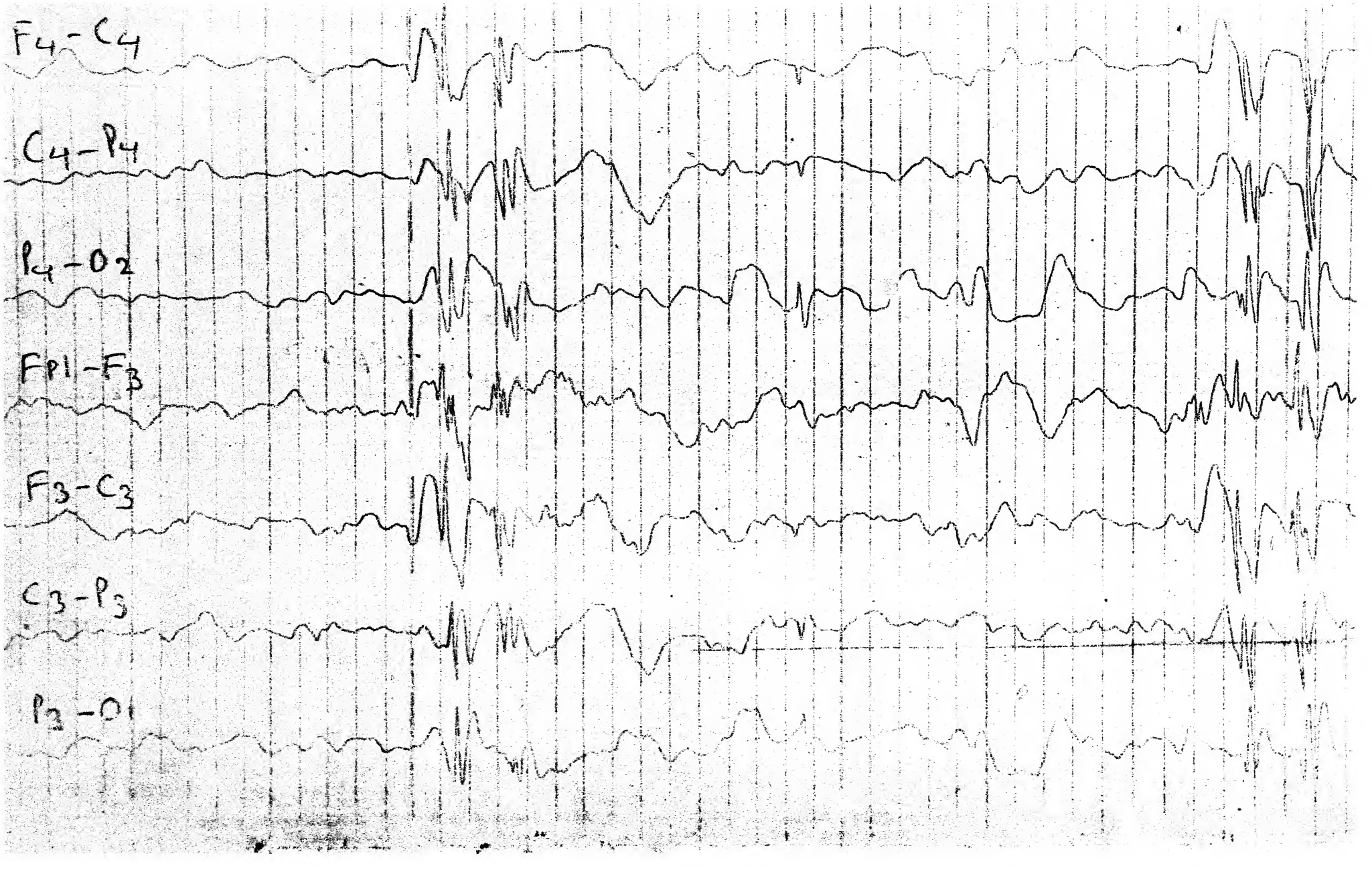
50 mv
1 sec.


F8 - A2
T4 - A2
T6 - A2
F1 - A1
F7 - A1
T3 - A1
T5 - A1

EEG No. 169 : Showing left sided focus in temporal lobe epilepsy.

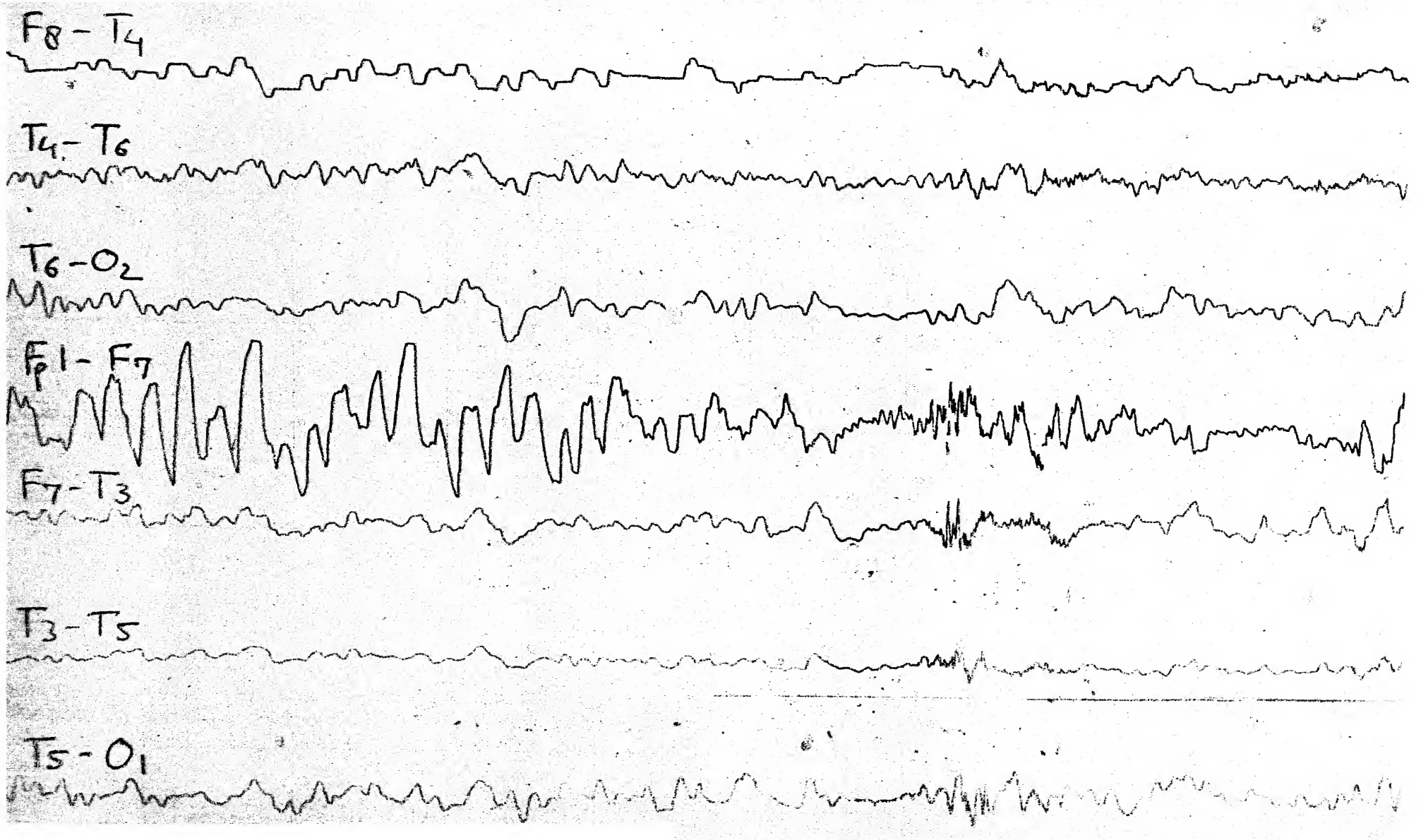

T6 - P4
P4 - Pz
Pz - P3
P3 - T5
T5 - A1
Fp2 - Fp1
O2 - O1

EEG No. 201 : Showing myoclonic epilepsy.

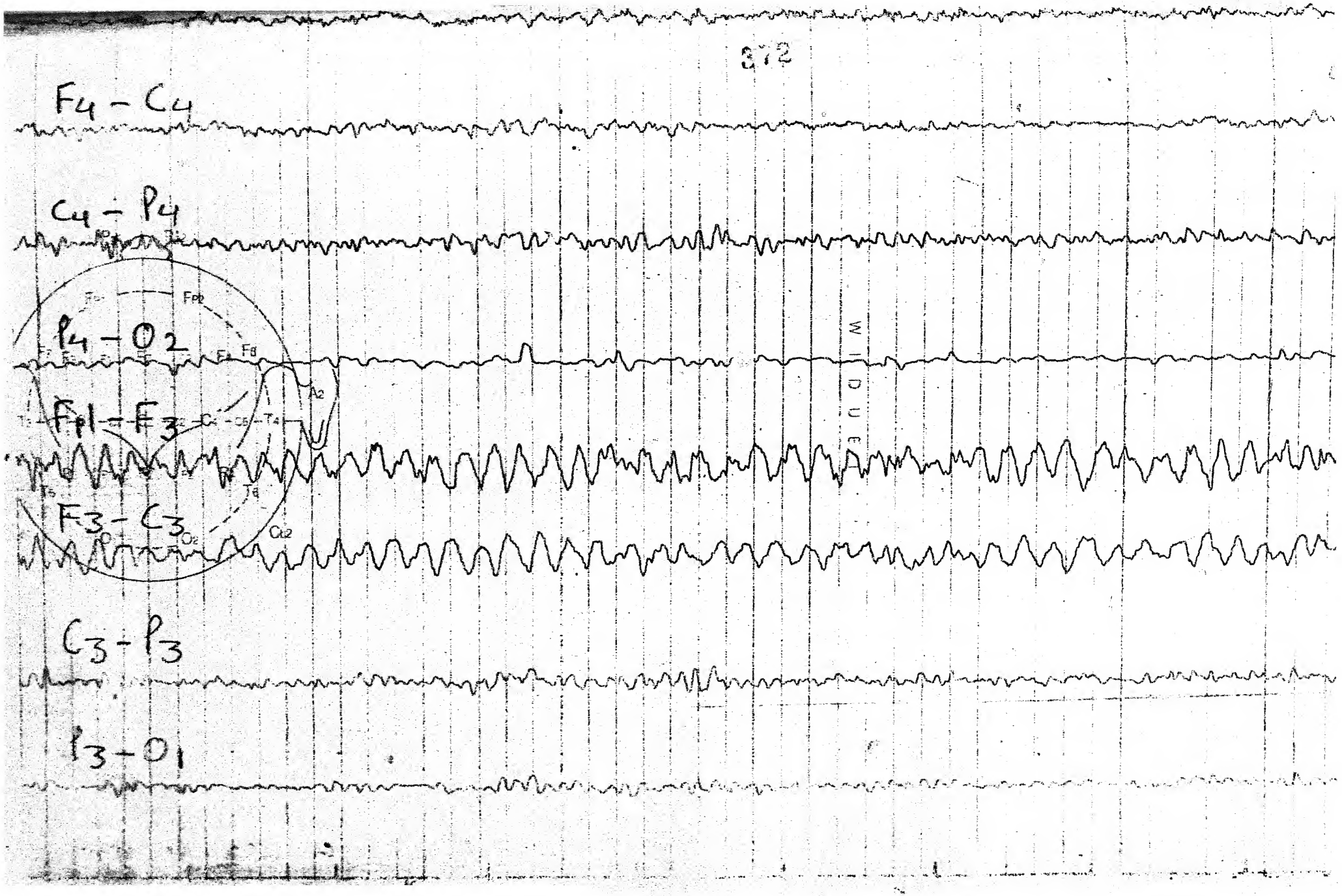

F4-C4
C4-P4
P4-O2
Fp1-F3
F3-C3
C3-P3
P3-O1

EEG No. 251 : Showing frontally predominant slowing indicative of generalized epilepsy.

50 MV
1 sec.


F8 - T4
T4 - T6
T6 - O2
Fp1 - F7
F7 - T3
T3 - T5
T5 - O1

EEG No. 321 : Showing slowing in frontal area in A case indicating intracranial space occupying lesion.


F4 - C4
C4 - P4
P4 - O2
Fp1 - F3
F3 - C3
C3 - P3
P3 - O1
1 mV
1 sec

ECG No. 321 : Same record showing slowing in C Rhm.

50 mv |_____
1 sec.

F8 - F4

F4 - F2

F2 - F3

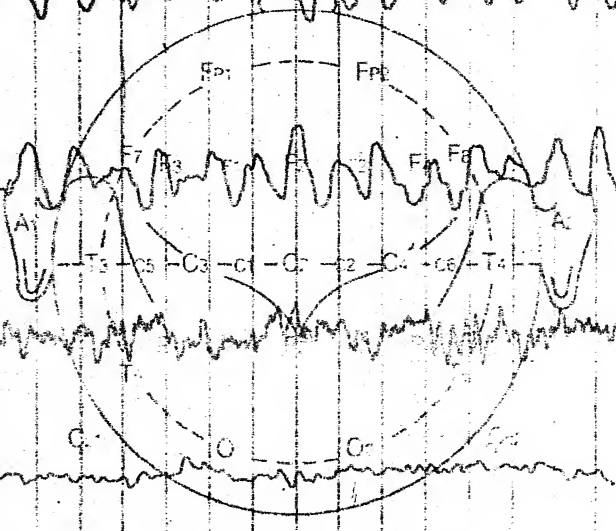
F3 - F7

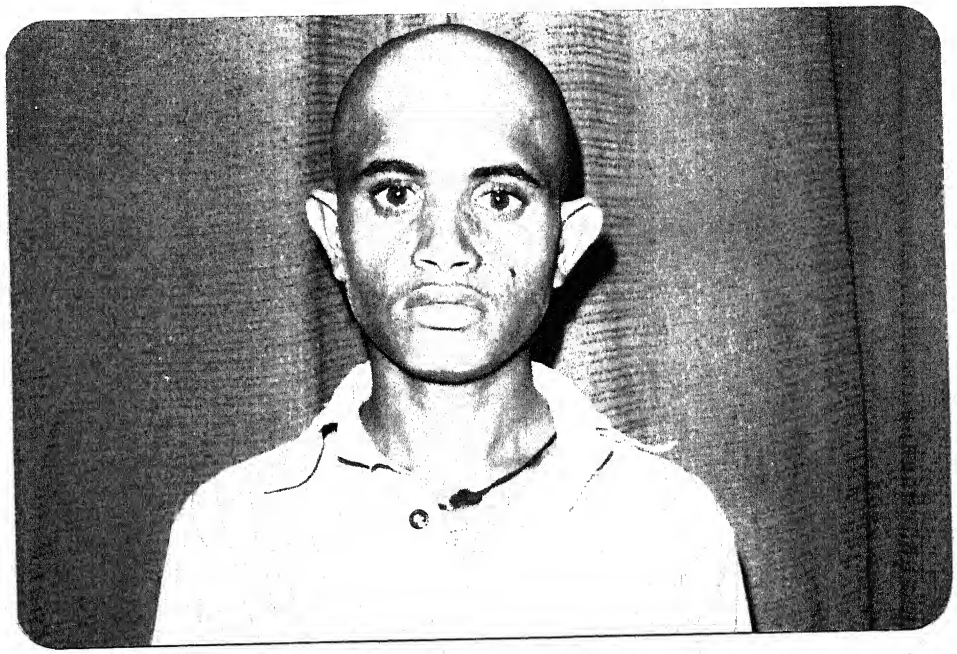
T4 - C4

C4 - C2

C2 - C3

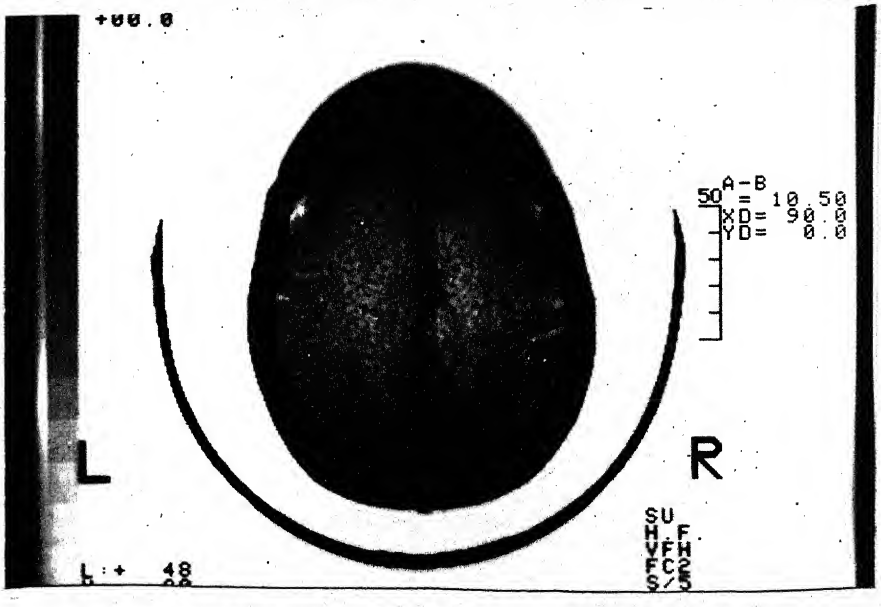
C3 - T3





A boy showing adenoma sebaceum on face, (tuberous sclerosis) presented with generalised seizures.

CT Scan head showing parietal lobe granuloma, 46 years old patient presented with generalised seizures.



CT Scan head showing parietal lobe granuloma in 30 years old patient presented with generalised seizures.

B I B L I O G R A P H Y

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APPENDIX - I

DEFINITION OF WAVES

EEG frequency bands - the rhythmicity of EEG signals gives a means of quantitatively describing EEG records, because the frequency of a rhythm can be measured EEG frequencies are conveniently classified into the following ranges or bands :

Delta - less than 4 Hz

Theta - 4 to less than 8 Hz

Alpha - 8 to 13 Hz

Beta - more than 13 Hz

Alpha frequency and alpha rhythm - Although frequencies in the range of 8-13 Hz are referred to as alpha, the true alpha rhythm as defined by Chatrian et al (1974) has the additional properties of being, 'Most prominent in the posterior areas, present most markedly when the eyes are closed and disappears on eye opening.

SPECIFIC WAVE FORMS

K Complex

A transient complex wave form consisting of slow waves sometimes associated with sharp components and often followed by a sequence of waves of about 14 Hz. The amplitude is very variable but usually about 200 Hv (Roth, Shaw and Green, 1956).

Spike

A transient wave, clearly distinguished from background activity, with pointed peak at conventional paperseeds and a duration of from 20-80 milli seconds.

Sharp Wave

A transient clearly distinguished from background activity with pointed peak at conventional paper seeds and duration of 80-200 milli seconds.

Spike and Wave Rhythm

A sequence of surface - negative slow waves usually with a frequency of 2.5 to 3.5 Hz having a spike associated with each wave. Sometimes these are several spikes in each complex which is then called a polyspike and wave complex. The amplitude may attain 1000 microvolts (Chatrian, Somasundaram and Tassinari, 1969).

Sleep Spindle

An episodic rhythm at about 14 Hz maximal over the frontocentral regions occurring during certain stages of sleep. The amplitude varies upto about 50 Hz.

Vertex Waves

A sharp potential maximal at the vertex negative in relation to other areas, occurring apparently spontaneously during sleep or in response to a sensory stimulus during sleep or wakefulness. The amplitude is very variable, but may attain 300 microvolts in children during sleep (Gastaut, 1953).

DESCRIBING THE EEG RECORD

Before beginning to describe an EEG record it should first be looked through quickly and a mental note made of the major features. The record is then described in chronological sequence in terms of the following features :-

1. The most persistent rhythmical feature - this might be the alpha rhythm.
2. Other rhythmical features, such as - delta, theta or beta rhythms.
3. Describe features of relatively long duration such as - an episode of spike and wave activity.
4. Discrete features of relatively short duration, such as isolated spikes or sharp waves.
5. The activity remaining when all the previous features have been described - sometimes called the background activity.

APPENDIX - II

EEG ELECTRODES AND ELECTRODE PLACEMENT

ELECTRODES

Electrodes are used to make connection between the conducting fluid of the tissue in which the electrical activity is generated and the input circuit of the amplifier. Types of electrodes are scalp electrodes, sphenoidal electrodes, nasopharyngeal electrodes, electrocorticographic electrodes and intracerebral electrodes.

Scalp electrodes are of following types - the pad electrode is made of silver rod belled out at the end and padded with sponge. Metal disc or cups are commonly used. They are attached to the scalp with an adhesive. Needle electrodes of platinum alloy or stainless steel are sometimes used (but have inferior recording characteristic).

ELECTRODE PLACEMENT

The majority of laboratories use the electrode placement recommended by the International Federation of Societies for Electroencephalography and Clinical Neurophysiology known as the 10-20 system. The initial description was given by Jasper (1958). It is stated that "Anatomical studies should be carried out to determine the cortical areas most likely to be found beneath each of the standard electrode positions in the average subject. The 10-20 system is based upon measurements from and standard points on the head, the nasion, the inion and the left and

right pre-auricular points. Two other points are also present F_{pz} and O_z. The position of all the electrodes are marked by a skin marking pencil prior to their application. The measurements are made with a tape measure or pliable rule as follows :

1. Measure the distance from Nasion to inion along the midline through the vertex and make a preliminary mark at the mid point Cz.
2. This is also the midpoint between the line drawn between the preauricular points(i.e. just anterior to the tragua).
3. Reapply the tape along the midline through Cz and mark points at 10, 20, 20, 20 and 10% of the total nasion - inion distance. These are positions of Fpz, Fz, Cz Pz and Oz.
4. Reapply the tape transversely through Cz and mark points at 10, 20, 20, 20, 20 and 10% of the total distance between the pre-auricular points. These are the positions of T3, C3, C2, C4 and T4. Note that the odd numbered positions are always on the left.
5. Measure the distance between Fpz and Oz by applying the tape along the great circle passing through - T3 and mark points at 10, 20, 20, 20, 20 and 10% of this length. These are the positions of Fp1, F7, T3, T5 and O1.

6. Repeat this procedure on the right side and mark the positions of Fpz, F8, T4, T6 and O2.
7. Measure the distance between Fp1 and O1 by applying the tape along the great circle passing through C3 and marks points at 25% intervals. These give the positions of F3, C3 and P3.
8. Repeat this procedure on the right side and mark the positions of F4, C4 and P4.
9. Check that F7, F3, Fz, F4 and F8 are equidistant by applying the tape transversely along the great circle passing through F7, Fz and F8.
10. Check that T5, P3, Pz, Pz P4 and T6 are equidistant in a similar manner.

WORKING PROFORMA

CLINICAL AND ELECTROENCEPHALOGRAPHIC STUDY OF EPILEPSY IN BUNDELKHAND

Case No.

OPD/MRD No. EEG No.

1. Name : 2. Age/Sex :

3. Occupation : 4. Marital Status:

5. Address : 6. Date of contact:

7. Age when Seizure started(in years) :

0 - 5
6 - 10
11 - 20
21 - 30
31 - 40
41 - 50
50+

8. Frequency of Seizures : Yearly: Monthly :
Weekly: Daily :
More than once a day :

9. Predisposing and Aetiological Factors :

Not known
Febrile convulsions
Birth injury with Anoxia
Inflammatory brain disease
Vascular lesions
Head injury
Intra-cranial space occupying lesions

10. Family History of Epilepsy :

Negative : Positive(Specify):

11. Precipitating factors :

Sleep
Sleep Deprivation
Fatigue
Light
Menstruation/pregeancy
Exposure to heat &/or cold
Alcohol
Emotional upset
Any other

13. Clinical Features

I. Type of Seizure :

A. Generalized : Tonic -clonic
Tonic
Atonic
Absence(Petit mal)
Atypical Absence
Myoclonic

B. Partial(Focal)

a. Simple partial -(without impairement of consciousness)

b. Complex partial-(with impairement of consciousness)

C. Partial Seizure Secondarily generalized.

D. Unclassifiable :

II. Tonic-clonic Seizures(Details of events)

a. Preconvulsive symptoms :
- Irritability/Depression/Abnormal feeling
- Related to head/giddiness/sudden myoclonic
- Twiches/others

b. The Aura - Sensory
- Psycho-sensory
- Emotional
- Autonomic

c. The convulsions :
- Epileptic Cry
- Consciousness
- Tonic spasm
- Clonic phase

d. Post convulsive phase :
- Consciousness
- Headache
- Mental symptoms
- Neurological deficit

III. Detailed Description of Seizures other than tonic-clonic Seizures.

Drug History :

14. Physical Examination

A. Neurological Examination

a. Higher Psychological Functions.

b. Cranial Nerves :

c. Motor System :

d. Sensory System :

15. Investigations

Blood - VDRL (Reactive/Nonreactive)

- Blood sugar :

- Serum Calcium :

X-ray : Skull

X-ray : Chest PA View :

Fundus Examination :

C.S.F. Examination : Normal/Abnormal

E.E.G. : Normal

Abnormal - Spikes
- Sharpwaves
- Slow waves
- Spikes and waves
- Polyspikes and waves
- Polyspikes
- Phase reversals
- Constant/paroxysmal

CAT Scan :

Drug Treatment :